THE

VETERINARY JOURNAL

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FEBRUARY, 1940

Editorial

TUBERCULOSIS

TUBERCULOSIS in domesticated animals is a subject which must continue to receive the attention of members of the veterinary profession in all parts of the world because of its prevalence and the necessity for its control. Work must include detailed researches into types, lesions, effects of the different types on different animals, variation of types, etc., as well as the production of the most efficient types of tuberculin for the diagnosis of the disease, especially in cattle. Further, the control of tuberculosis in the herds of countries must continue to be pursued as far as it can possibly be carried out consistent with the facilities available. The research work of Nielsen and Plum published in THE VETERINARY JOURNAL last month is evidence of the fact that yet another method of infecting cattle with the bovine type of tuberculosis may be more common than is generally realised. It will not, of course, be denied that the great bulk of tuberculosis infection in cattle is transmitted to healthy from already infected bovines, but in examining for apparent breakdowns in herds, the human being affected with bovine tuberculosis must be considered as a possible source.

In the present number we publish part of a general account of the

pathology and pathogenesis of tuberculosis in domesticated animals compared with man and it is hoped to follow this article by others dealing in turn with the various domesticated animals. It is only comparatively recently that the differences of lesions in some of the domesticated animals have been studied and this has enabled a true estimate of the incidence of the disease to be made in some of the animals concerned.

The control of tuberculosis must always be dependent primarily on the diagnosis of the disease and that, in turn, involves the use of tuberculin which will detect the infected animal in the greatest possible number of cases. This is not the place nor the time to discuss tuberculins, but it may be pointed out that in this and other countries research work must continue till it results in the evolving of a product which will be found efficient to the maximum degree in the hands of all users.

The schemes in operation in this country for the eradication of tuberculosis are, for the present, unfortunately, to be limited. It is evident from the views expressed by the farming community that this decision is viewed with some disappointment. The keen interest taken by the farming community throughout the country in the schemes and their progress is evidence of the success attached to them. Though, naturally, difficulties arise from time to time and all may not see eye to eye in some of the conditions demanded, the fact that there is a demand for their continuation on an increasing scale by the stock owners is proof of their effectiveness, at least, in some districts. It is unfortunate that present-day conditions must interfere with their extension. It is to be hoped that, when conditions again become normal, the schemes will extend to their greatest capacity and that the age-old question of the disposal of the "reactor" will again receive consideration and be suitably settled.

EMERITUS PROFESSOR JOHN SHARE JONES,

M.D. (V., h.c.), D.V.Sc., F.R.C.V.S., M.Sc.

It is with a considerable amount of pleasure that we reproduce an account of the ceremony when a presentation was made to Emeritus Professor J. Share Jones by the University of Liverpool on the occasion of his retirement from the Directorship of Veterinary Studies and the Chair of Veterinary Anatomy in the University.

An active man, whose main interest during his long life has been the advancement of his profession, has, alas, reached the age when he has had to give up his official appointments to make room for younger men who will, we feel assured, follow in the path which he has so carefully and laboriously

prepared and shaped. The name of Share Jones will always be associated with the School of Veterinary Science of the University of Liverpool for it has been largely under his guidance and rule that its present position has been attained. His interests, however, have lain and, we doubt not, will continue to lie far beyond the School itself, for he has the wide subject of veterinary science and its various ramifications so very much at heart. We cannot believe that Share Jones will simply go into retirement, though he richly deserves a well-earned rest after 35 years of activity in the University. Those who know him well cannot imagine that the veterinary world has lost one of its important champions by his retirement from the University. No! We look forward to hearing much of him and his activities in the future and we know that any new venture in which he may be found will assuredly have the interests of his profession as its one and only object.

We extend to him our very hearty congratulations on the position he has attained and the very great respect in which he is held as shown by a perusal of the proceedings published in this volume, and wish him a further long, useful spell of life when he may devote himself to the accomplishments of ambitions which we know he may be cherishing, unhampered by official duties. He has accomplished much—he will accomplish more!

To his wife, Dr. Mary Share Jones, our warmest good wishes are also extended.

THE NEW EDITORSHIP OF "THE VETERINARY JOURNAL"

It was with much pleasure that we read the Editorial published in *The Veterinary Record* of February 10, 1940, which deals with The Veterinary Journal and its newly-appointed editors. We have the interests of the veterinary profession very much at heart and it will be our endeavour to maintain the standard of the publications in this journal so that the profession shall at all times have brought to its notice recent observations which may be applied to the maintenance of health and prevention and treatment of disease among our domestic animals. The Veterinary Journal during most of its life has catered more for the practitioner than for members of the profession engaged in other aspects of veterinary science and it is the full intention of the present editors to continue on these lines. We are deeply grateful to *The Veterinary Record* for giving prominence to this recent change of editorship and we thoroughly agree that there is ample scope for the healthy existence of both journals.

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General Articles

THE PATHOLOGY AND PATHOGENESIS OF TUBERCULOSIS IN DOMESTICATED ANIMALS COMPARED WITH MAN

By J. R. M. INNES,
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Introduction

It is perhaps not always realised that there are still unsolved problems associated with the pathology and pathogenesis of tuberculosis. The variations in the reaction of different animals and man to the tubercle bacillus must be fully explained if pathology is to have that broad comparative basis so essential for its future development.

In English veterinary literature there are no papers comparable to the many excellent English and American publications dealing with the medical aspect of the disease. It is hoped that a paper, such as this, with suitable illustrations, will offer a background for those who wish to view the disease through the eyes of the comparative pathologist. For these reasons the article has been written more in the manner of a critical annotation.

The first part deals with a general consideration of the reaction of tissue to the tubercle bacillus, the pathogenesis of the disease in man and a section on the disease in dogs and cats; a second part will deal with bovine tuberculosis. The literature is not intended to be complete and is given in a "directional" form suggesting the most important publications, some of which are of historical interest.

Tuberculosis is a chronic disease process in which, even in one animal species, the anatomical variations may appear to be many, but if it is considered as a whole in different animals and man, they might seem almost endless. These variations are related to the inter-action of the destruction and repair of tissues, the factors which influence the end result being (a) the size of the infecting dose of bacilli, (b) the virulence of the latter, (c) the resistance of the host, (d) the state of allergy, (e) the path of infection, and (f) in animals particularly, the type of bacillus is of great importance. Although all are contributory, not one of these factors alone is decisive in establishing the pathogenesis of the disease. These are statements often made in the literature, but we still lack accurate information of their influence in any given tuberculous infection. As a result certain lesions have been interpreted differently, and there is still much controversy of the rôle of allergy and immunity in the development of the disease.

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The pathogenesis of tuberculosis in man and animals can be understood only when we can answer accurately the questions (a) how does the bacillus enter the body?; (b) what is the immediate and subsequent reaction of the host to one or more infections?; and (c) how does the infection spread through the body? These problems have only been partly solved.

It is necessary to review modern concepts of the disease in man as recent studies of animal tuberculosis have been constructed on a comparative basis. As far as the subject concerns the veterinarian recent work in animals has been mainly that of Nieberle and his pupils in Germany.

Effect of the Tubercle Bacilli on Tissues

It is an old observation that (a) there are differences in reaction of similar hosts to different strains of the same organism, and (b) there are similar differences in reaction when animals of the same species are infected by a type of bacillus of standard virulence. According to Rich, virulence is irrevocably linked with resistance, hence to a consideration of the former must always be appended the question " for what animal? "

With these reservations the entrance of tubercle bacilli into a tissue may be regarded as evoking a standardised reaction which soon reaches the macroscopic stage of a small nodule (tubercle). (In some animals, e.g., dog and cat, this is not strictly accurate.) The lesion in the initial stages is essentially the same whether the organism is of high or low virulence or even of a non-virulent character such as BCG — all will cause a fundamentally identical tubercle. The cellular response has been regarded by Rich as the most primitive organised type of reaction in which a vascular inflammatory mechanism is not involved and which occurs before the onset of any tissue destruction.

The bacilli, having entered the tissue, are immediately surrounded by small numbers of polymorphonuclear leucocytes and lymphocytes (Fig. 1). Soon, however, the cellular focus enlarges by an accumulation of phagocytic cells—the epithelioid cells, at which stage the leucocytes may be scanty or absent (Figs. 3 and 4).

There has been much controversy about the nature and genesis of these epithelioid cells. They are usually large, closely packed, round or polygonal cells with indistinct margins with abundant eosinophilic granular, or sometimes clear, cytoplasm, and with a small vesicular nucleus which may be at one pole of the cell (Figs. 21-24). The genesis of these cells has been worked out by Sabin, Doan, Forkner, Cunningham and others. Their immediate precursors are the mononuclear phagocytes or macrophages which are identical with the blood monocytes and which are continually wandering from the blood into tissues. By the use of vital staining it has been shown that the epithelioid cell in common with the blood monocyte possesses a cytoplasmic rosette. These macrophages or mononuclear phagocytes have been given many different names at various times, e.g., endothelial leucocytes (Mallory) said

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to be derived from vascular endothelium, adventitial cells (Marchand), clasmatocytes (Ranvier), polyblasts or amœboid wandering cells (Maximow), histiocytes (Aschoff). These cells which give rise to the epithelioid cells of the tubercle are to be found in many different diseases and localities, and are referred to under other names, e.g., as scavenger cells in the nervous system following tissue destruction and known as compound granular corpuscles or *gemästete Glia*—probably microglial in origin (Fig. 24); as "heart failure" cells or alveolar phagocytes in the lungs (Fig. 22); and as macrophages in and around abscesses.

Tubercles will form in this manner either in intra- or extravascular locations, indeed they appear to form in any tissue in which mononuclear phagocytes are present, even in tissue culture. At a later stage lymphocytes appear more numerous and may form a thin layer peripheral to the epithelioid cells, (Figs. 4-7). Subsequently in man and in most species of animal, there are formed, either by fusion of several epithelioid cells or by amitotic nuclear division, the characteristic multinucleated giant cells, first described by Langhans. These may be of different shape and size and their numerous nuclei are often, but not always, arranged in a horseshoe fashion around the periphery of the cells (Figs. 8-20). Giant cells have been regarded as a post-necrotic development, but they are often found in areas totally unrelated to necrosis. They appear to be a response to the need for the phagocytosis of foreign material; the lipoid fraction of the tubercle bacilli is said to contain a maturation factor necessary for their genesis.

Silver impregnation methods can demonstrate a delicate framework of argyrophil fibrils ramifying throughout the tubercle even at an early stage of development (Fig. 25). The relationship of these fibrils to collagen is disputable, but there is good reason for believing that they represent simply a pre-collagenous stage.

The focus can now be regarded as a fully formed tubercle (Figs. 5, 6 and 7) which is characteristic, at least, for certain stages of the disease, in man and some animals; similar lesions can be provoked by non-virulent bacilli, e.g., BCG (Figs. 1-5), although the fate of the lesion may be very different. Epithelioid and giant cell lesions can also be produced experimentally by subcutaneous injection of olive oil, droplets of mercury, suspensions of agar, etc. (see Cunningham and Tomkins); the "tubercle" is not, therefore, specific for the pathogenic bacillus.

The subsequent course of the lesion varies greatly in natural and experimental conditions, and may be summarised as follows:

(1) If the numbers of bacilli are small, they may fail to propagate and/or they may die rapidly; the lesion may then resolve completely illustrating a potentiality of tissue reversion from altered character to normal which is insufficiently stressed. This must occur before connective tissue develops otherwise scar formation will result. Resolution of this kind may be commonly

seen following the intravenous injection of an organism such as BCG into rabbits, and must also occur during the natural disease.

(2) By fibroblastic proliferation and the production of collagen, the tubercle may be converted into a hyalinised mass and/or be encapsulated by fibrous tissue, while calcification may occur in the centre, which in human primary infection may form a locus for later ossification. This lesion might be regarded as obsolete or healed (Figs. 26, 28, 29, 37 and 38).

(3) The centre of the tubercle may undergo necrosis and subsequent caseation (Figs. 5, 7 and 37). The bacilli by themselves have little power to cause necrosis in the unsensitised body, nor, as was once thought, is necrosis due to the non-vascularity of the tubercle. The supposedly non-vascular nature of the tubercle is often emphasised, but as cells appear to wander in from neighbouring vessels, tissue fluid may well do likewise, otherwise all tubercles would suffer the same fate of tissue death. The development of necrosis appears to be influenced partly by the number of bacilli present and partly by the development of sensitisation of the body to the tuberculo-proteins. It is now known that of the chemical fractions isolated from tubercle bacilli, it is the lipoids which produce tubercle formation and the proteins which cause acute inflammation and tissue death. Central necrosis occurs before fibrosis and encapsulation and liquefaction may subsequently ensue.

(4) The necrobiotic process may extend centrifugally to destroy fibrous tissue as soon as it forms, in which case fresh infections into adjacent normal tissue will form contiguous or satellite tubercles (Figs. 34 and 35).

(5) Bacilli may enter the blood or lymph stream and be transported to organs near to, or distant from, the primary source and thus give rise to secondary lesions in which the above processes may be repeated.

(6) An acute fulminating inflammatory reaction may occur in response to massive infection (or reinfection) in which the intensity of tissue destruction overwhelms the "defensive" cellular mechanism.

The body, therefore, responds to infection by the production of a tubercle (Fig. 7) or by acute exudative inflammation (Fig. 32); hence, the terms "proliferative" and "exudative" inflammation, now commonly in use, and under certain circumstances either process may predominate. Factors which control this variation have been mentioned, but allergy (in the sense of Pirquet-Ranke) requires further consideration. The effects of allergy in the body is seen in the well-known Koch phenomenon. If a healthy guinea-pig is inoculated subcutaneously with tubercle bacilli of low virulence, a nodule forms which breaks down, does not heal, and the regional glands become enlarged and caseous; this is followed by generalisation of the disease and death in about 2-3 months. The effect of inoculation of a guinea-pig already infected is markedly different. A local swelling appears in a few days which ulcerates but soon heals, and the regional glands are not enlarged. This does not mean that the glands are not infected, but that the bacilli are mostly retained at the site of inoculation, and/or die in great numbers, or those which

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do reach the glands find them unfavourable for free propagation. This fixation of bacilli to the site of entry is the result of an acute exudative inflammation which to some degree is characteristic of a second infection; the nodular tubercle, on the other hand, is more characteristic of a first infection. These phenomena are observed in natural disease of man and animals depending on whether it is a primary infection or a secondary (re)infection (Figs. 30, 31, 32 and 33). These facts must not, however, be accepted too strictly as it has been shown, particularly by Rich, that exceptions occur and that allergy must be separated from immunity. Rich contends that, although allergy and immunity may be coincidental, they are not identical as suggested by Krause and others. Following infection the body acquires an altered reactivity towards the bacillus, but both the normal animal, following a single infection, and the allergic animal may respond either by tubercle formation or by exudative inflammation. Which type of reaction occurs is primarily dependent on the size of the infecting dose and where the bacilli lodge. The difference is seen in the greater tendency of the allergic animal to react by exudative inflammation, i.e., with more extensive damage and tissue death. As a result of previous infection an animal is allergic to the products of the bacillus and the response is necrosis and inflammation, but there is also a development of acquired resistance which localises the bacilli to the site of entry where they may not thrive, or where the spread of viable organisms is delayed.

Tuberculosis in Man

Parrot (1867) stated that the portal of entry of the tubercle bacillus is indicated by the lesions in the regional lymph nodes, a dictum which has withstood the test of all subsequent work. As an index of primary infection the lesion must be the only lesion in the body or the oldest, and must be in a situation which is a possible portal of entry for the bacillus from outside sources. The lymph node draining the path of such a primary infection always shows a lesion, and together with the primary infection forms the so-called "primary complex" of Ranke (Figs. 34, 35, 36, 39 and 53). Particularly in mucous membranes and/or if the number of bacilli are small, entry may be "silent" and only the regional lymph node appears (at any rate macroscopically) affected; this is often referred to as the "incomplete primary complex."

Behring stated that the disease in the adult was the end stage of a childhood infection, and Ranke subsequently believed that tuberculosis was comparable to syphilis with (a) a primary infection period, (b) a secondary anaphylactic stage and metastatic spread, and (c) a tertiary stage of increased resistance expressed by limitation of the disease to the lungs.

Pulmonary tuberculosis (phthisis) of the adult is not the first manifestation of the disease but is the result of (endogenous or exogenous) reinfection. In many individuals, primary infection occurs in infancy, but in most the lesions heal, calcify and may even ossify. In a few the lesion progresses,

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and the child dies, while in others infection spreads by direct extension, by the natural passages and by hæmatogenous and lymphogenous paths. Primary infection occurs in nearly every living person of civilised communities.* The path of infection in about 80 per cent. is *via* the respiratory tract, in about 15 per cent. *via* the intestine, and the remaining 5 per cent. probably comprise sites such as the nasopharynx, skin and middle ear. These figures vary in different countries; for example, in Scotland infection by the alimentary tract is probably much higher. Blacklock's figures are: primary thoracic infection, 61.1 per cent., and primary abdominal infection 35.7 per cent.; the recent data of MacGregor and Alexander are very similar.†

Primary pulmonary infection may occur as early as the first month after birth, usually in the first six years, and less frequently after ten years. Opie gives the incidence as 43 per cent. of children up to five years, 80 to 90 per cent. of those over 20, and nearly 100 per cent. of persons over 30 years.

The entry of the bacilli causes a caseating broncho-pneumonic focus which is small and sharply demarcated from adjacent normal lung (Fig. 35). The lesion may occur in any part of the lung, is usually single, and often, but not always, sub-pleural, and is the so-called Ghon lesion, although Kuss was the first to describe it (Figs. 34, 35 and 36). It is always accompanied by a lesion in a regional lymph node which can be related anatomically to the lung focus (Figs. 34, 35 and 36). The spread of the bacilli from lung to lymph node is probably by means of the macrophages which abound in all advanced tuberculous lesions. In most cases these lesions heal by fibrosis, hyalinisation and calcification, and the lung lesion, in time, even by ossification (Fig. 38). These healed forms constitute a valuable index of the first infection of an individual, and may be found in cases of chronic (adult) phthisis. Relatively rarely in children this primary infection does not heal, the caseous pneumonic process spreads through the lungs and death may result from an acute tuberculous caseous pneumonia (Fig. 41).

The mode of infection to the lung has been contested since Behring stated that the bacilli passed through the alimentary mucosa and mesenteric lymph nodes without producing any lesions, to settle eventually in the lungs—views

* In autopsies on infants and children carried out by Blacklock in Glasgow, healed calcified lesions were not found. This is in contrast to the findings of continental, American and other British workers (Aschoff, Rich, Opie, Pagel, etc.). Blacklock states that nearly all the children in this district of Scotland when infected with tuberculous lesions of the lungs and/or tracheo-bronchial nodes die in childhood, and that he was unable to trace any obsolete calcified pulmonary foci in adults. He concludes that there must be a low racial resistance to infection in Scottish children. The higher incidence of abdominal infection must be due to the higher number of infections by the bovine type.

† "It must be clearly recognised that a consideration only of clinically manifest pulmonary tuberculosis in children gives a misleading conception of the real importance and frequency of primary tuberculosis lung lesions. The fact that they are often clinically undetectable and that illness and death may be due to secondary or metastatic manifestations of the disease have tended to obscure the truth that the lungs play a dominant role as a portal of entry and as a primary infection site of all ages of children."

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which were later supported by Calmette. The evidence, however, is open to severe criticism and Blacklock, for example, states that no definite conclusions can be drawn regarding this mode of infection where tuberculosis affects only, or is most advanced in, the lungs. All available modern data indicate that primary pulmonary lesions are due to direct inhalation infection *via* the air passages.

As a result of primary infection, a state of allergy in the sense of Pirquet-Ranke, i.e., hypersensitiveness, develops. The lesion in the lymph node may progress, with infection of adjacent nodes, to produce enlarged glands—the so-called scrofula of olden times. By exacerbation of the primary focus, the individual enters the post-primary phase of generalisation. This is due to the entrance of bacilli into the blood stream with spread of the infection to a few or many different organs of the body, the lungs usually being also involved in this secondary spread. In almost every individual post-primary lesions occur in the lungs, but the foci are abortive and may heal in the same way as the primary lesion. The Ghon focus is thus often surrounded by a ring of "satellite" tubercles (Figs. 34 and 35). These lesions may progress and produce an anatomical form different from that of chronic adult pulmonary tuberculosis, "punched out" thin-walled cavities (Fig. 46) and cortico-pleural lesions being part of this process. The lesions in the visceral organs which may arise by blood spread may also be progressive—constituting the chronic post-primary disseminated tuberculosis (Fig. 47). Various bones may be affected, usually in the vicinity of the metaphysis, with production of tuberculous osteomyelitis, periostitis and arthritis; the uterus in the female, the testicle, prostate, seminal vesicles in the male, one or both kidneys, ureter and bladder, the brain and the suprarenal glands are other organs which are commonly affected. In all, the subsequent course is the same, viz., progressive caseation with contiguity spread of the infection, while the lymph nodes draining the organs concerned may also become involved. Invasion of the blood stream by a large number of bacilli may cause a fatal acute miliary tuberculosis (Fig. 43), affecting perhaps every organ in the body. (Particular mention must be made of the involvement of the brain in this post-primary stage. Isolated foci form in the brain which may caseate and rupture into the sub-arachnoid spaces or ventricles to produce the tuberculous meningitis with intense exudative reaction so characteristic in young people. Rich states that this is the process constantly invoked in tuberculous meningitis, and that the meninges are never affected by direct hæmatogenous spread, an idea supported by the recent work of MacGregor and Green.)

Chronic miliary tuberculosis (Fig. 45), chronic proliferative lesions in the lymph glands, skin lesions, and tuberculosis of the special sense organs are other forms of the disease which come into this category of post-primary dissemination.

The special features of "first infection" or childhood type of tuberculosis thus include the inconspicuous nature of the primary lesions, the early

and pronounced affection of the lymphatic system and the tendency to extensive and often fatal infection *via* the blood stream.

In later life some adults, who have had a previous primary infection, develop tuberculosis which is limited to the lungs. It was once thought that an individual with a primary infection could not be reinfected from without, but opinions have changed, and adult phthisis is now said to be due to a superimposed new or exogenous (re)infection. In any case, the bacilli appear to be arrested in the lung to form a lesion distinct from that of the primary infection. The (re)infection (Puhl's) focus (Figs. 48 and 49) is commonly situated in the apical lobe, and changes of the regional lymph nodes do not occur, or are represented only by microscopic abortive tubercles (contrast with the primary complex). In a series of routine autopsies all transitions from such fresh apical lesions to fibrous scars (healed lesions) or to progressive phthisis can be followed. The apical lesions and the insignificant involvement of the lymph nodes indicate an acquired resistance. Many individuals are reinfected in this way, but relatively few develop the progressive fatal disease. This lesion may also heal by fibrosis and calcification, but rarely by ossification. If it does not heal progressive consumption of the adult develops. The spread of the disease within the lungs then takes place largely by bronchial aspiration, the resultant anatomical changes depending on this manner of spread as well as the virulence of the bacilli and host resistance. If the infective dose is massive or especially virulent, and/or resistance is low, breakdown and liquefaction of the caseous focus may occur with discharge into a bronchus, and cavity formation may result (Fig. 51). At first lobular in character, the lesion may assume lobar proportions in which all stages of progress of the disease can be seen. Erosion of blood vessels or development of small aneurysms may lead to severe or fatal hæmoptysis; the overlying pleura may be thickened (Fig. 52) and may show signs of an active pleurisy, and pneumothorax may be an additional complication. In the more chronic type, productive inflammation predominates, and fibrosis and thick-walled cavities are an outstanding feature (Fig. 52). Bacilli may be coughed up to reach the mouth and nasopharynx and produce tertiary lesions in the tonsils, pharynx and larynx, and if swallowed may cause intestinal ulceration. The process in chronic phthisis is, therefore, mainly localised to the lungs, all other lesions being simply appendicular to the bronchial system. As Pagel says, it seems paradoxical that adult phthisis, which is so often fatal, should be considered an expression of immunity, but the latter indicates simply the restriction of a process, which would be otherwise disseminated, to an organ that is most susceptible. Acute miliary tuberculosis rarely occurs at this stage in contra-distinction to its frequency in the post-primary dissemination phase. In the adult the bacilli, which must escape into the blood stream, even in large numbers, must reach only resistant tissue; many, no doubt, die while others produce abortive tubercles. A heavy enough seeding of the blood stream by viable organisms will always, however, result in acute miliary tuberculosis.

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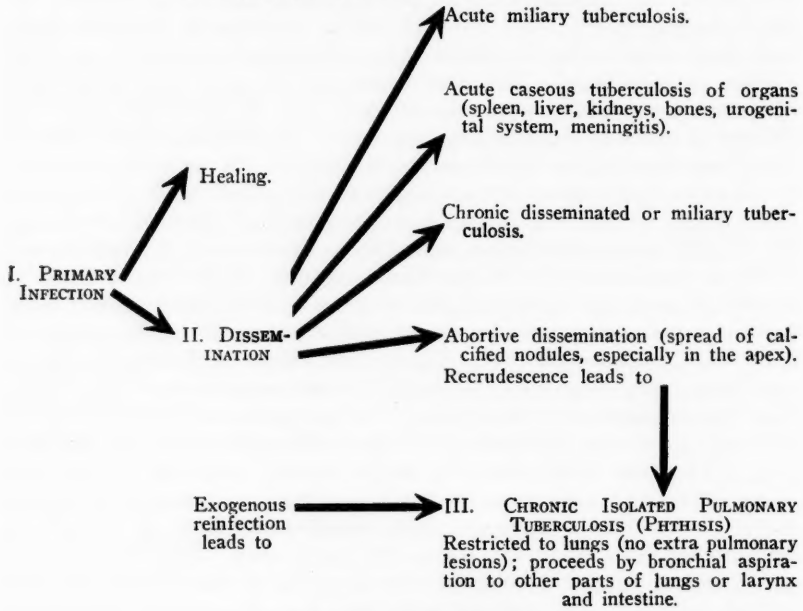
Summary

Tuberculosis in the human can be separated into different phases, all of which are intimately related; primary infection is the initial stage of the evolution of the disease which may heal, remain quiescent or which may pass into the second stage of dissemination which may be acute, chronic or abortive. The third stage, due to reinfection of the adult body, is characterised by the appearance of a chronic isolated pulmonary tuberculosis.

DIAGRAM OF THE EVOLUTION OF THE DIFFERENT STAGES OF TUBERCULOSIS IN MAN

(According to Kayne, Pagel and O'Shaughnessy)


Acute miliary tuberculosis.
Acute caseous tuberculosis of organs (spleen, liver, kidneys, bones, urogenital system, meningitis).
Healing.
Chronic disseminated or miliary tuberculosis.
I. Primary Infection
II. Dissemination
Abortive dissemination (spread of calcified nodules, especially in the apex).
Recrudescence leads to
Exogenous reinfection leads to
III. Chronic Isolated Pulmonary Tuberculosis (Phthisis)
Restricted to lungs (no extra pulmonary lesions); proceeds by bronchial aspiration to other parts of lungs or larynx and intestine.

Explanation of Figures

The legends contain additional information to that in the text. Figs. 1-29 illustrate the development and fate of the tubercle; Figs. 1-7 show the series of changes in the formation of the tubercle as seen in the lungs of rabbits following the intravenous injection of BCG, the animals being killed at different intervals. The latter are not necessarily indicative of the periods required for the various cellular changes to develop nor does the lesion develop constantly in this way during the course of the spontaneous disease. Figs. 34-52 illustrate the different phases of the disease in man, and the remainder are from cases in the dog and cat.

(To be continued.)

PLATE I

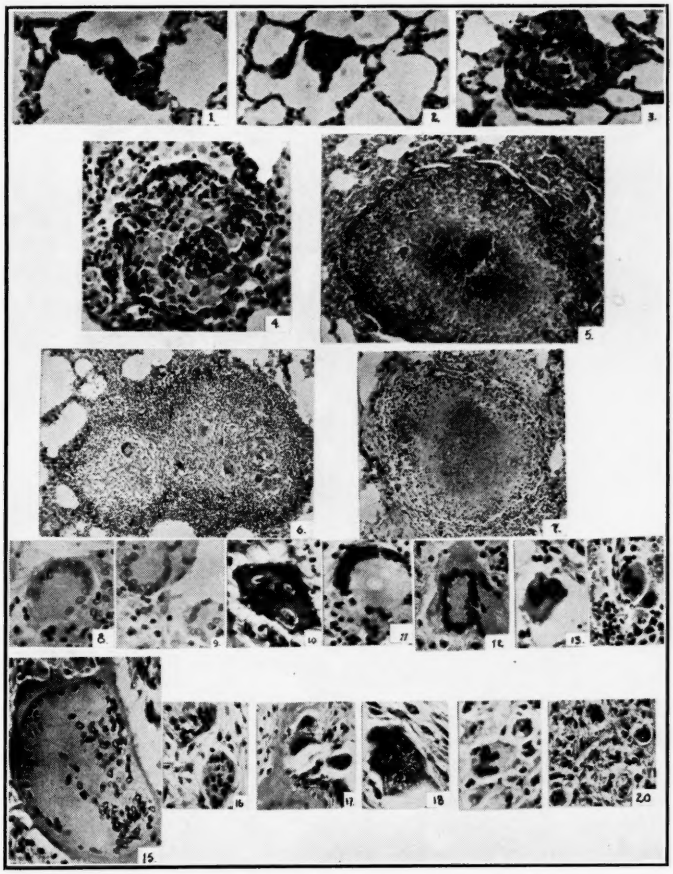


FIG. 1.—Lung. Rabbit. Killed 24 hours after the injection of BCG. Showing the initial intra-alveolar focus of polymorphs and lymphocytes around a clump of bacilli; the latter naturally cannot be seen in this section; no alveolar exudation. H.E.

FIG. 2.—Lung. Rabbit. Killed 48 hours after the injection of BCG. The focus is larger and projects into the alveolus; most of the cells present are lymphocytes. H.E.

FIG. 3.—Lung. Rabbit. Killed 6 days after injection of BCG. The lesion is larger and shows a central area of necrosis around which are large faintly staining cells—early formation of epithelioid cells. H.E.

FIG. 4.—Lung. Rabbit. Killed 21 days after injection of BCG. The focus is now a clearly demarcated nodule visible macroscopically and consists of a conglomerate mass of epithelioid cells around a minute central area of cell debris, and a thin peripheral zone of lymphocytes. Many of these tubercles produced by an avirulent strain such as BCG probably do not progress beyond this stage but resolve completely. H.E.

FIG. 5.—Lung. Rabbit. Killed 200 days after injection of BCG. Showing a large well-developed tubercle with extensive necrosis and caseation; the zones of lymphocytes and epithelioid cells are distinct, but no giant cells are present. H.E.

FIG. 6 & 7.—Tuberculosis. Compare the previous figures with these tubercles from (6) miliary tuberculosis, lung, child, in which the lesion is more proliferative in type without central caseation but with giant cells, epithelioid cells and lymphocytes, and (7) miliary tuberculosis, lung, horse, in which central caseation is marked.

FIGS. 8-20.—Giant cells. These show the variations to be found in the form and size of the tuberculous giant cells and the number and distribution of the nuclei; some conform to the so-called "typical" large, round giant-cell (Langhans) with the nuclei arranged in a concentric, horse-shoe fashion around the periphery, others are more atypical but are just as commonly found. 8—From tuberculous meningitis, calf; 9—from primary lung infection, calf; 10—from an intimal lesion, longitudinal sinus, monkey; 11—from udder, tuberculosis cow; 12—from miliary tuberculosis, lung, horse; 13—tuberculosis, tongue, human. Compare these giant cells with those found as: 13—A foreign body giant cell in a chronic inflammatory lesion; 14—a megakaryocyte, normal bone marrow; 16—an osteoclast concerned with normal bone resorption, rib, 1-day-old pig; 17—osteoclasts in excessive numbers, osteo-dystrophia fibrosa, pig; 18—a giant cell of the osteoclastoma or giant cell tumour of bone, man; 19—giant cells in a case of osteogenic sarcoma, man; 20—bi- and tri-nucleated giant cells of Hodgkin's disease, man (Dorothy Reed or Sternberg cells). H.E.

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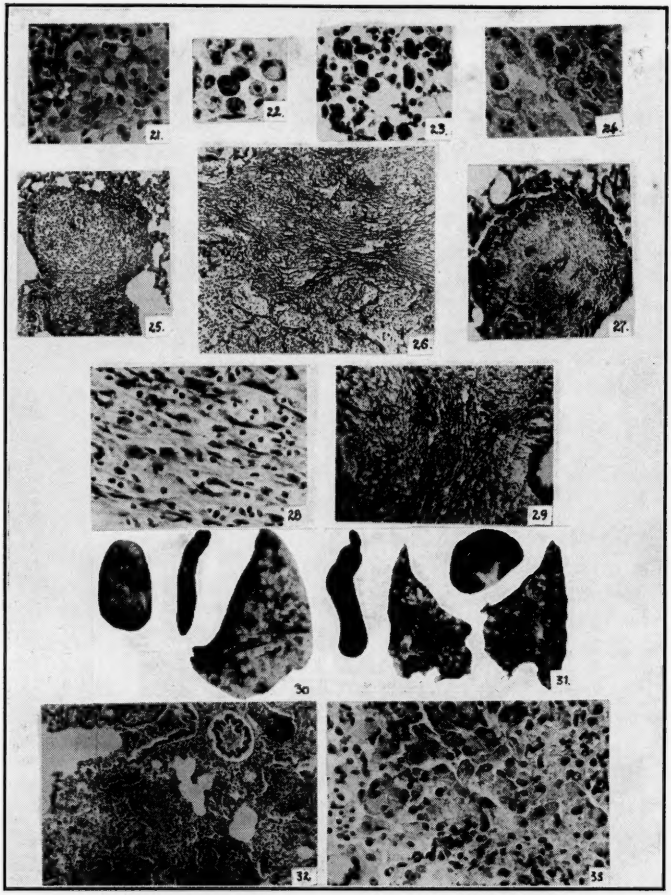
PLATE II



FIGS. 21-24.—These figs. show (21) epithelioid cells in a tubercle compared with other cells of the same nature arising in different situations and in response to stimuli other than the tubercle bacilli; (22) Alveolar phagocytes, lung, man (often called "heart failure cells"); (23) Macrophages in the sinus of a lymph node showing erythrophagocytosis from a node regional to a hæmorrhagic area in a suspected swine fever case; frequently erroneously termed hæmorrhagic lymph-adenitis; (24) Compound granular corpuscles (gemästete Glia) in an area of demyelination in the brain, Schilder's disease, man; the cells are large and round with eccentric nuclei, and contain much lipoid derived from the broken-down myelin.

FATE OF THE TUBERCLE

(For caseation see FIGS. 5 and 7, and for calcification and ossification see FIG. 38.)

FIG. 25.—Fibrosis. Lung, rabbit injected with BCG. Showing early fibrillary formation in a tubercle demonstrated by a silver impregnation reticulum stain; the fibrils form a delicate basket work at the periphery of the tubercle; these may persist even in advanced necrotic areas. Wilder's method.

FIG. 26.—Fibrosis. Lung, showing a more advanced state of fibril formation which form a closely anastomosing network. Wilder's method.

FIG. 27.—Tubercle, lung, child, stained for elastic fibres to show their persistence even in caseous areas. Weigert's Elastic stain.

FIGS. 28 & 29.—Fibrosis. Advanced healed fibrotic lesion, calf; showing the numerous fibroblasts in the midst of dense collagenous tissue, the latter being more clearly brought out in the silver impregnation. (FIG. 29.)

FIGS. 30 & 31.—*Reinfection.*—These figures show the effect of experimental reinfection in the rabbit. (30) Control rabbit injected intravenously with virulent tubercle bacilli; died after 50 days with acute miliary tuberculosis; the multiple nodular lesions in the lungs, spleen and kidney are illustrated. (31) Experimental rabbit injected with a virulent strain 70 days after previous avirulent infection with BCG, thus producing an *isolated* pulmonary tuberculosis with early cavitation in the infra-clavicular area; the spleen and kidneys are free from lesions. This process is comparable with chronic isolated pulmonary tuberculosis of man.

FIGS. 32 & 33.—Lung, rabbit. This animal was reinjected with BCG 21 days after the initial inoculation and died within 24 hours, after showing acute respiratory distress. This demonstrates the occasional high degree of allergy developed in some animals after one infection. Such an animal responds to the second injection by an intense pneumonic exudation of large phagocytic cells and fluid which may be sufficient to cause death. The effects are limited to the lungs. (32) Low power showing the extent of the alveolar exudation, and (33) high magnification showing the epithelioid-like cells in the alveoli.

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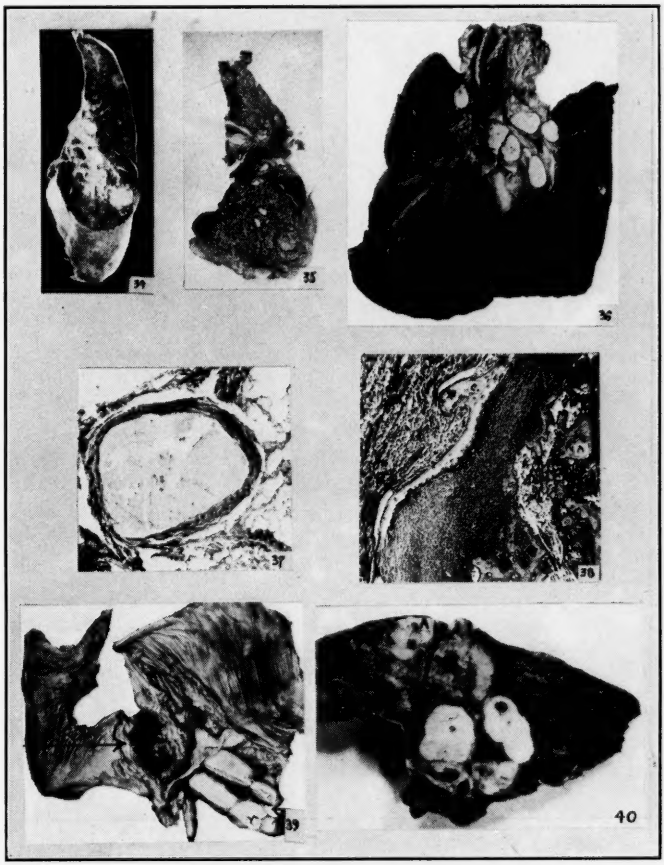
PLATE III



TUBERCULOSIS IN MAN

(Figs. 34-52 for comparison with the disease in the dog and cat.) Primary Infection in Children.

Fig. 34.—Primary lung complex. Primary (Ghon) focus in lower lobes; a caseous sub-pleural nodule with satellite tubercles around; the line of lymph spread infection is marked by a row of tubercles passing towards the root; two intra-pulmonary root glands are enlarged and caseous.

Fig. 35.—Early primary lung complex. Large section of the lung showing a primary focus in the lower lobe with early caseous enlargement of the root glands; small tubercles in the lymphatics form rows passing between the primary focus and glands.

Fig. 36.—Primary focus in left lower lobe (<—) as a small caseous sub-pleural nodule with satellite tubercles; gross caseous enlargement of root glands and inferior and superior tracheo-bronchial glands on left side and of one right superior tracheo-bronchial gland.

Fig. 37.—Primary focus showing the histology. A sub-pleural (pleura on the left) lobular, caseous, pneumonic area with complete encapsulation; the lung tissue adjacent shows no changes. If healing takes place, this focus becomes calcified and even ossified. (See next figure.)

Fig. 38.—Primary lung focus, showing a focus at a much later stage of development in which there is a calcified centre (A), a fibrotic capsule (B), and ossification of the latter (C). This is the fate of all obsolete primary pulmonary lesions in the human, but does not appear to occur in animals.

Fig. 39.—Primary abdominal complex, showing a primary ulcer at the end of the ileum with enlarged caseous regional lymph nodes.

Fig. 40.—Fresh primary complex with extensive caseous enlargement of the regional hilar glands; the Ghon focus (A) shows localised extension due to bronchial spread; for convenience, the photograph has been placed with the apex to the right.

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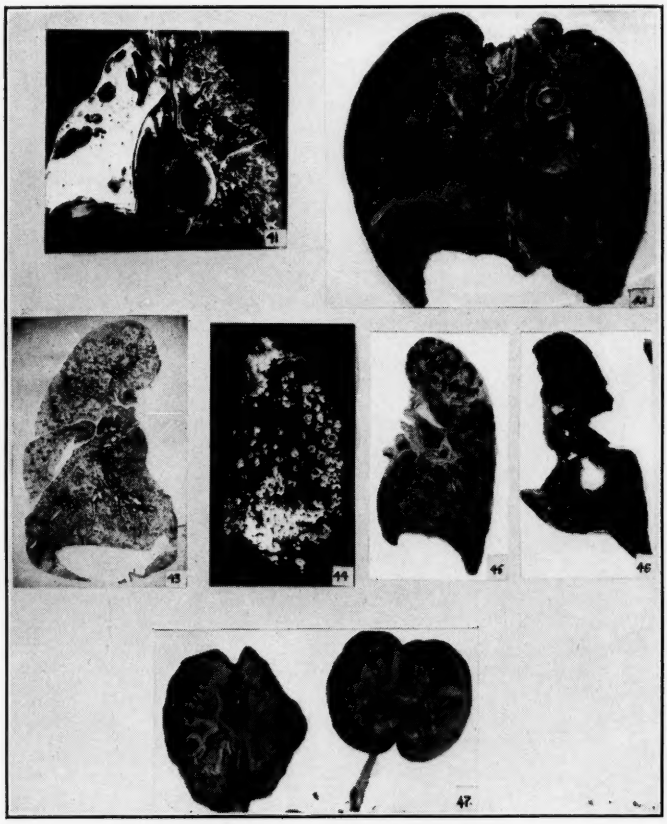
PLATE IV



POST-PRIMARY EXTENSION AND SPREAD

FIG. 41.—Lobar caseous pneumonia; unilateral extension of the infection by aspiration; early thin-walled cavity formation.

FIG. 42.—Acute miliary tuberculosis. The left lung is collapsed by pleurisy and is free from tubercles; the right side is studded by very minute tubercles, evenly scattered throughout; this involvement is but part of a general dissemination throughout the body, and in this case was universal.

FIG. 43.—Acute miliary tuberculosis. Large lung section. This shows the process more clearly; the characteristic lesions are minute tubercles seeded uniformly throughout the lung, in which caseation has hardly begun; again simply part of general dissemination throughout the body; the primary focus for this dissemination need not be pulmonary.

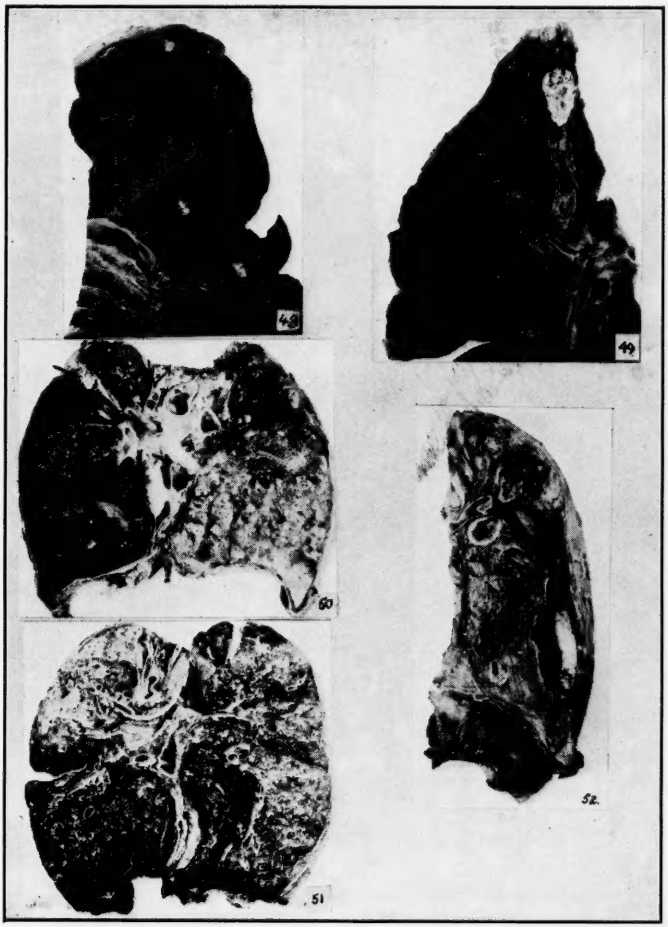
FIG. 44.—Lobular type of caseous pneumonia; the extension of the infection has arisen by the breaking down of a cavity and resultant aspiration.

FIG. 45.—Subacute or chronic miliary tuberculosis; this occurs when infection of the blood stream has not been severe enough to cause early death; the miliary lesions have thus time to develop more fully. The lungs are studded with numerous caseous foci, many of them being confluent; the lesions vary in size from a pin-head to half a centimetre. Other organs in the body may be similarly involved, but cavity formation is rare.

FIG. 46.—Thin-walled cavities. This picture shows the type of cavity which is found in the lungs when the latter are involved in a more chronic process at this stage of evolution of the disease. They appear as if they were "punched out," and have thin walls, and should be compared with FIGS. 52 and 59.

FIG. 47.—Chronic urogenital tuberculosis. Hæmatogenous spread following in the course of post-primary dissemination, causing a chronic caseating pyelo-nephritis, pyelitis and ureteritis. This is given as an example of the spread which occurs at this stage; other organs may be similarly infected.

PLATE V



CHRONIC ISOLATED PULMONARY TUBERCULOSIS OF THE ADULT DUE TO REINFECTION

FIG. 48.—Typical calcified reinfection (Puhl's) focus near the lung apex. These lesions may heal and give rise to the typical apical scars; if not, they form a nidus for all future developments and extensions of the disease to produce the chronic phthisis; the associated lymph node does not show any change.

FIG. 49.—Old, calcified, reinfection focus at the apex, with no involvement of the regional node.

FIG. 50.—Reinfection focus in right upper lobe; confluent, caseous pneumonia in left lower lobe, due to aspiration infection from a cavity which is not shown; the lesions in parts show the typical clover-leaf form ("acino-nodular").

FIG. 51.—Early cavitation of the focus in the right upper lobe with aspiration infection and the production of acino-nodular lesions.

FIG. 52.—Chronic phthisis. Showing extensive cavitation in the left lower lobe with thick fibrotic walls and thickening of the pleura. Compare with thin-walled cavities in FIG. 46.

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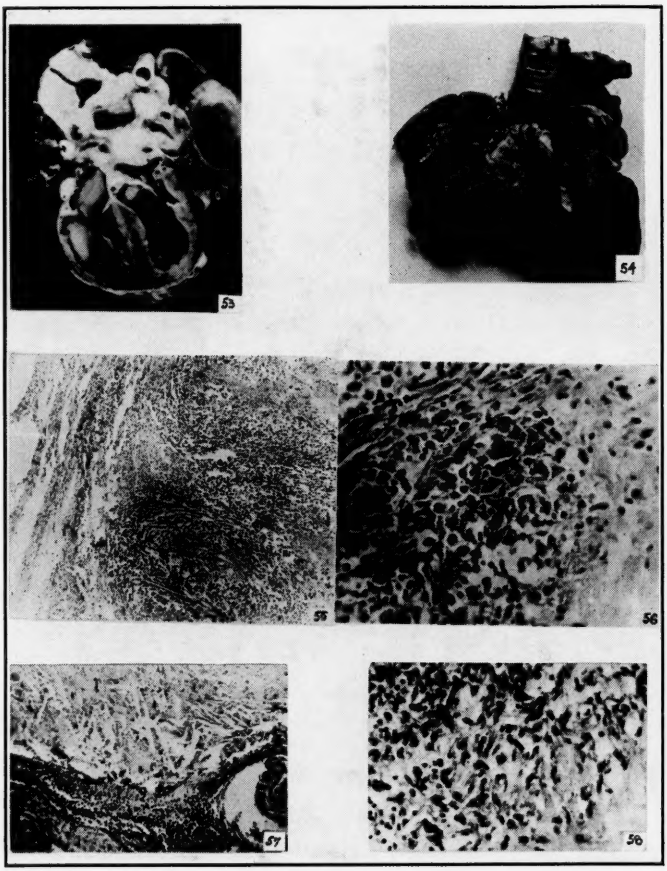
PLATE VI



TUBERCULOSIS IN THE DOG

FIG. 53.—Primary pulmonary infection. Transverse section through lungs and heart just in front of the bifurcation of the trachea. Showing a fully developed primary complex as a small hazel-nut-sized focus in the hilar region of the right apical lobe of the lung(←—); enlargement of the lymph nodes of the same side (A) and slight enlargement of the hilar glands on the other (B). This case also showed a few nodules scattered over the pleura and pericardium (C). Compare with the figs. showing similar lesions in children. Tuberculin negative. Terrier. M. 4 yrs.

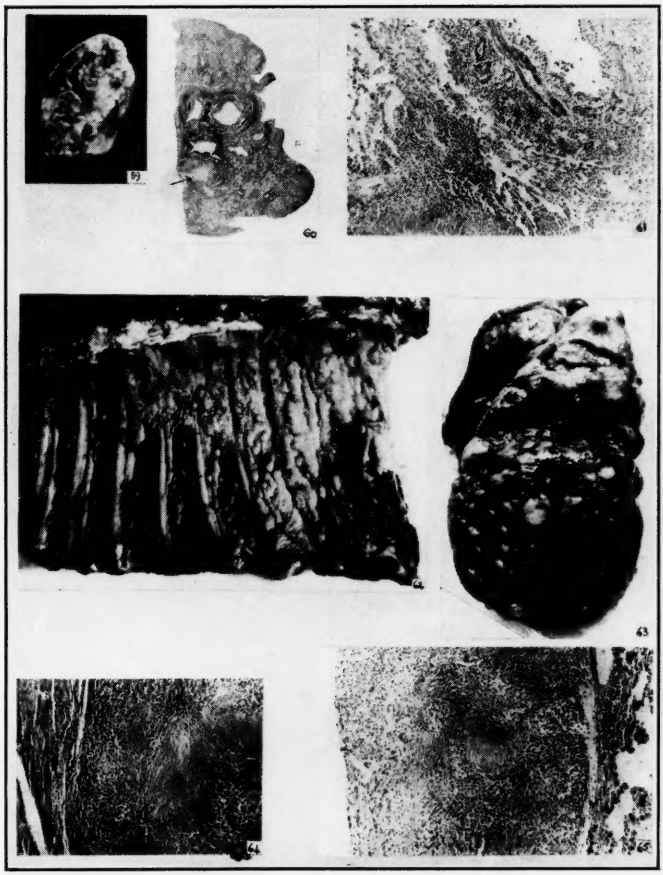
FIG. 54.—Primary pulmonary infection. Showing a primary complex in a more advanced condition, with massive caseous enlargement of the bifurcation (A) and right hilar nodes (B). The lung lesion was a large walnut-sized focus present in the dorsal border of the right cardiac lobe (C), which was partially excised for histological purposes; this focus is fused with the enlarged lymph glands; the left hilar node was free from lesions; no generalisation; bacilli numerous in smears. Bull terrier. M. 2 yrs.

FIG. 55.—Primary infection. Showing the lung lesions separated without any encapsulation from partially collapsed lung on the left. Irregular confluent foci of necrosis surrounded by a cellular infiltration consisting mainly of macrophages, lymphocytes and fibroblasts, the latter arranged in a trabecular manner. No typical tubercle formation; no giant cells or calcification. Same case as FIG. 54.

FIG. 56.—Primary pulmonary infection, showing a higher magnification of the cellular infiltration in which macrophages, fibroblasts and lymphocytes can be clearly distinguished. Same case as FIG. 55.

FIGS. 57 & 58.—Primary abdominal infection. Incomplete primary complex. Mesenteric lymph node from a dog which was killed because of carcinoma testis. The node was not enlarged and showed a small bean-sized caseous focus in the centre; there were no other tuberculous lesions in the body. Bacilli were present in smears but not recovered by culture; the lesion might thus be regarded as quiescent or obsolete. Histologically, the lesion consisted of a central necrotic area with a few calcific deposits, but without any definite fibrotic capsule; between the normal gland structure and necrosis there is remarkably little reaction; a few macrophages and fibroblasts lie amidst collagenous strands; no giant cells. FIG. 57.—Low magnification of the lesions with normal lymph gland below. FIG. 58.—High magnification of an area at the edge of the lesion.

PLATE VII



POST-PRIMARY DISSEMINATION

FIG. 59.—Primary pulmonary infection with extension by aspiration throughout the lung, producing bronchitis, caseous pneumonia, and bronchiectatic cavitation; the irregular cavity is not comparable to the thin-walled "punched-out" cavities of childhood tuberculosis. Naked-eye photo of transverse section through one lobe of the lung.

FIG. 60.—Large section from same case as FIG. 59, showing the multiple caseous foci mostly in relation to bronchioles and part of the cavity (→).

FIG. 61.—Same case as FIGS. 59 & 60, showing the peribronchial infiltration and the edge of a caseous area at bottom left of photo; the cellular reaction is similar to that seen in FIG. 56.

FIGS. 62 & 63.—Pleurisy and pericarditis. This shows the appearance of the nodular lesions which occur on the pleura and may involve the pericardium (FIG. 63) and other thoracic structures such as aorta, but the changes may be more diffuse; sero-sanguinous transudate may be so abundant as to cause compression atelectasis.

FIGS. 64 & 65.—Pleurisy. Showing the lesions present in the costal pleura (64) and the lungs (65). The histological picture is similar to that seen in the primary lung lesion, although it may be more granulomatous. This is from a case of primary pulmonary infection which showed similar pleural involvement to that seen in FIG. 62.

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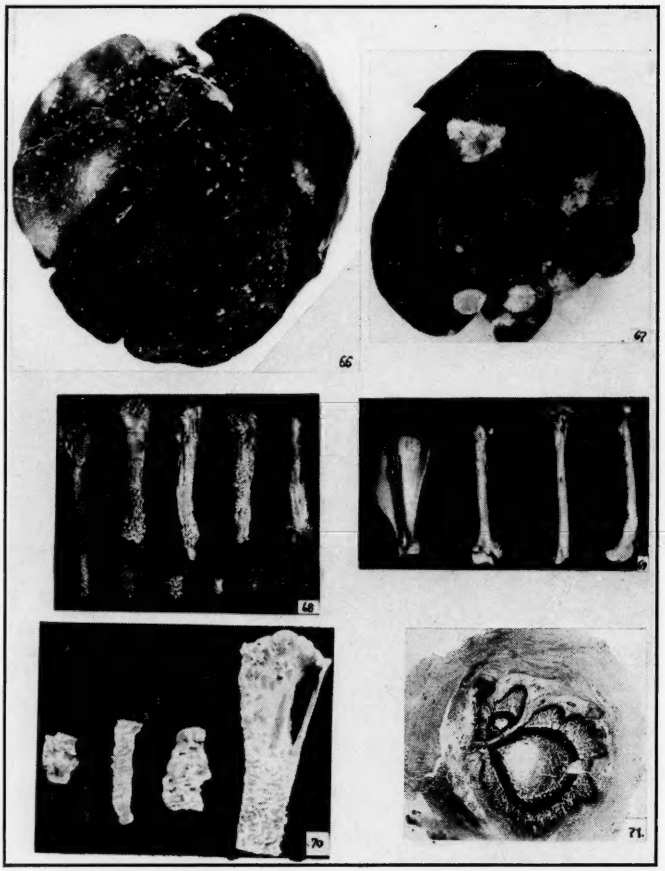
PLATE VIII



FIGS. 66 & 67.—Liver. Subacute miliary tuberculosis. Showing types of dissemination; FIG. 66.—The whole organ is evenly and diffusely scattered by small tubercles, while in FIG. 67 the lesions form isolated large nodular masses. Both would show similar histological picture to that seen in other canine lesions.

FIGS. 68-71.—Acropachia or hypertrophic osteo-periostitis. FIG. 68.—The limb bones and digits from a case of pulmonary tuberculosis; all the bones are covered by an exuberant periosteal overgrowth. FIG. 70.—Another case showing similar lesions in the tibia, tarsal and metatarsal bones, but all bones in the leg (right) which were examined were similarly affected; this is from the same case as FIG. 59, in which the probable duration of the disease was about 1 yr. FIG. 71.—Photograph of whole transverse section (slightly enlarged) of the tibia from same case as FIG. 70, showing the formation of excessive spongy bone arising from the periosteum to form another irregular layer of compact bone; note also the massive paraosteal fibrosis. FIG. 69.—Early lesions of limb bones of dog with carcinoma involving head of pancreas with secondary extension to the liver. No tuberculosis.

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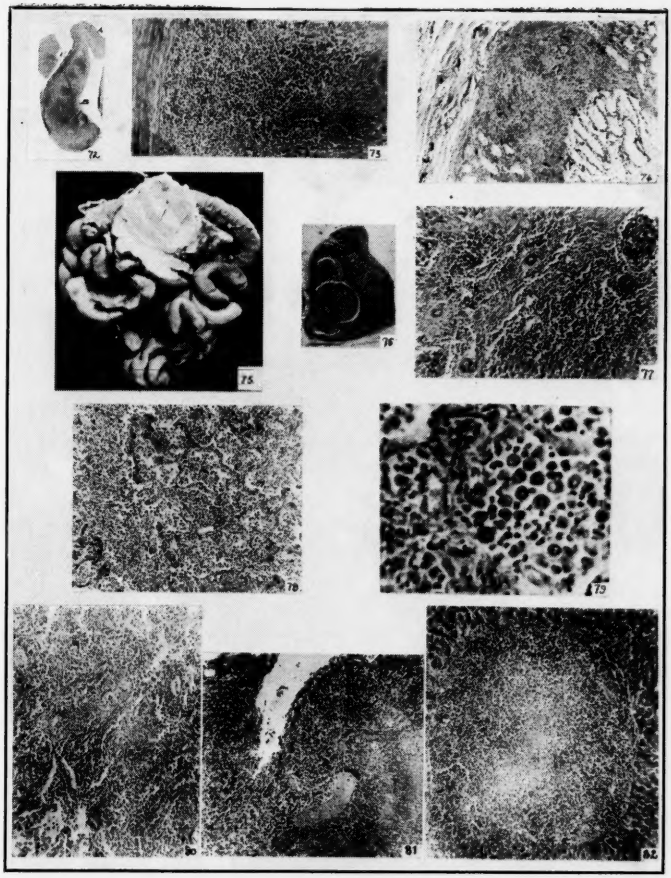
PLATE IX



TUBERCULOSIS OF THE CAT

FIGS. 72 & 73.—Primary abdominal infection—complete complex. A single isolated 1 cm. ulcer was present in the mucosa of the terminal part of the ileum with gross caseous enlargement of the regional mesenteric lymph nodes; dissemination to the lungs with the production of caseous pneumonia, isolated foci in the spleen and liver. This section shows normal mucosa at (A) and ulceration of the thickened mucosa at (B) extending deep into the muscle; the cellular infiltration at the base of the ulcer (FIG. 73) adjacent to the muscle consists mainly of large macrophages and lymphocytes; there are no giant cells. The serosa was fused with a small lymph node (C). The histology is identical to that seen in the canine lesions; bacilli were numerous in smears and the lesion was obviously a progressive one.

FIG. 74.—Primary abdominal infection—complete complex; this shows a small fibrous scar in the mucosa of the ileum in a region which was fused to a large caseous mesenteric node such as is seen in FIG. 75. This might be regarded as a healed minute primary intestinal focus even although spread of the infection had occurred to the regional nodes and to the lungs.

FIGS. 75 & 77.—Primary abdominal infection—incomplete complex; showing a massively enlarged caseous mesenteric lymph node; in this case there was no ulceration visible macroscopically in the intestine; isolated foci were present in the lungs, liver, spleen and kidney. FIG. 77.—The lesion shows a similar picture to that seen in canine tuberculosis, the cellular reaction consisting mainly of macrophages and lymphocytes; no giant cells or calcification.

FIG. 76.—Primary abdominal infection—"quiescent incomplete complex." The only lesion present in this animal was a small caseous area in a mesenteric lymph node similar to the dog case, FIG. 57. This section shows a caseous area on the left (A), sharply demarcated from normal lymph node by a thick well-developed fibrotic capsule (<—); bacilli were present in smears but could not be recovered culturally or by guinea pig inoculation.

FIGS. 78 & 79.—Tuberculous pneumonia. Early generalisation to the lungs from a primary abdominal infection with the production of an acute pneumonic process involving whole lobes; the lungs were enlarged, voluminous and consolidated; the alveoli are filled with a cellular exudate consisting mainly of macrophages (alveolar phagocytes), lymphocytes and a few polymorphs; these cells are seen in greater detail in the high magnification (FIG. 79).

FIGS. 80 & 81.—Subacute miliary tuberculosis. Lungs; low magnification of a caseous area (FIG. 80), an area adjacent to a bronchiole and with the same type of cellular reaction as seen above. (FIG. 81). Much exudation of large macrophages in the adjacent lung alveoli.

FIG. 82.—Subacute miliary tuberculosis in the liver; the naked-eye picture was similar to FIG. 66. Low magnification of a nodule showing central necrosis and cellular reaction as seen in other canine and feline lesions.

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MOULDY HAY A CAUSE OF BROKEN WIND IN HORSES AND ABORTION IN COWS AND MARES

By EDWARD MORGAN, F.R.C.V.S., D.V.H.
Venezuela.

THE fact that in a certain tropical country where hay is not harvested one does not see a case of broken wind in horses supports the opinion of farmers around my home in Cardiganshire that mouldy hay is the principal cause of broken wind—chronic alveolar emphysema. The horses in Venezuela, with the exception of a few, graze throughout the year in the open, but when needed for hard work they are fed on whole maize (Indian corn) along with the green food. Great care is taken that the animals are watered prior to and not soon following a feed of maize, thus avoiding colic and a ruptured stomach.

During the last thirty years in North Cardiganshire the occurrence of broken wind in horses exists to a very small extent compared with former years. This coincides with and may be largely due to the modern system of storing hay in up-to-date buildings. Years ago many of the farmers stored hay within badly ventilated old stone or mud-walled buildings, with the result that the hay bordering on the walls was covered with grey-coloured mould. To-day almost every farm has a proper hay shed, i.e., a roofed building consisting of only posts or pillars on the sides, hence the hay is well ventilated and is found free from mould. In one particular farm where a great number of horses were stabled during the winter it was very rare to find a horse over the age of five years free of broken wind. Since the system of storing hay in modern hay sheds has been adopted the horses are sound in wind. Lately, while discussing this particular question with several farmers, they came to the conclusion that since mouldy hay has not been fed to the horses broken wind is rarely seen. Professor Wm. Williams dealt with broken wind under the section of Dietetic Diseases.

Mouldy Hay as a Cause of Abortion in Mares and Cows

There is much evidence that mouldy hay is the cause of abortion in these animals. I shall only mention one case out of hundreds which have been witnessed. This case occurred last month. On a farm in Cardiganshire a farmer was obliged to deposit a few loads of hay within the walls of an old-fashioned damp barn where it became mouldy. As the writer had advised the man in charge not to feed any pregnant stock with this hay, he did not

exactly do so, but instead he thought he might use it as a bedding for them, one of which was a fine in-foal shire mare. Although the mare had abundance of well-harvested food in front of her, yet she would sample the bedding. Next morning the fœtus was found on the stable floor. Fortunately the mare felt little the worse as far as illness was concerned, but it was a loss and disappointment to the owner to lose what might have been one day a valuable foal, due to a simple oversight on the part of the caretaker.

Our knowledge of this fungus (grey mould) on hay is meagre. The fungi —Claviseps purpurea or Ergot of Rye as well as Corn Smut—are classed as Ecbolics.

Bansse—*vide* THE VETERINARY JOURNAL, 1903, p. 80—signalises the effects of mouldy clover on the horse, observing delirium, with staring eyes, perspiration and foaming, followed by weakness, loss of appetite, loss of power of hind legs, and muscular tremors of elbow and thigh.

In Veterinary Toxicology, Lander mentions the effects of the mould oidium (on bread) on horses recorded by Perrin in THE VETERINARY JOURNAL, 1907, p. 248. There were internal rumblings, frequent defæcation and passage of small quantities of urine, difficulty in moving, membranes injected, pulse 60 full and hard. Later the animal fell. The pulse was weak, there was coma followed by vertigo and death, after repeated seizures.

In the numerous cases which have come under our observation of horses and cattle fed on mouldy hay, especially in mares and cows, we have observed but few of these symptoms. The principal symptoms are broken wind, discharge from the nostils of a greyish white colour, abortion, poor condition, pendulous abdomen, staring coat, paroxysm of coughing, especially after a liberal drink of water, and at the same time flatus is expelled through the anus.

Gamgee describes an epizootic disease in Scotland due to mouldy grass which produced acute stomach and intestinal trouble, cerebral derangement and paralysis. Acute stomach tympany has been observed in the horse as the result of mouldy grass.

Saccharomyces farciminosus is a pathological organism which produces epizootic lymphangitis in horses.

Mucor mucedo Linnæus is very common, living in organic substances in decomposition, for instance, horse dung. It is the cause of a deadly disease in bees, the so-called " Mucorine " or May's disease.

Mucor pusillus is often found in bread which has been soaked in water. It is very pathogenic to rabbits. It has only been found in man in a case of otomycosis by Jakowski.

Lichtheimia ramosa.—This also has been observed by Jakowski in a case of otomycosis. According to Vuillemin it is frequently found in the nasal mucosa of horses.

Rhizomucor parasiticus.—A mycelium at first greyish, later brownish. It is pathogenic for guinea-pigs and rabbits. It was found in the expectoration

of a woman by Lucet. The patient had been considered at first to be suffering from tuberculosis. She recovered under a potassium iodine and arsenical treatment.

An affection due to Aspergillus fumigatus is very common in some parts of France among pigeon breeders (gaveurs de pigeons). The same affection attacks the pigeons. It is characterised by the presence of mycotic nodules in the lungs, liver, kidneys and other organs. The infection is probably caused by spores of A. fumigatus being present in the grain used for feeding the pigeons.

Botrytis bassiana causes the disease of silkworm called muscardin. Quevedo has described an aspergillus which he believes to be the cause of a type of encephalo-myelitis in horses in South America. He calls the organism A. mayidis.

FOWL TYPHOID IN SCOTLAND

By J. E. WILSON

Royal (Dick) Veterinary College, Edinburgh.

It is generally believed that in Great Britain fowl typhoid (an acute infectious disease of fowls caused by Bacterium gallinarum) is more or less confined to Wales and the contiguous counties of England, although from time to time sporadic outbreaks occur elsewhere.

My experience tended to confirm that view, for after carrying out a large number of post-mortem examinations of fowls from all parts of Scotland during the period 1927-1936, only two outbreaks were confirmed.

In 1936 it was brought to my notice that a disease had been prevalent for many years on the island of Lewis (Outer Hebrides), causing heavy losses and possessing symptoms suggestive of fowl typhoid. It is believed by the inhabitants that this disease, which is known in Lewis as Galair na Cearc (Gaelic—the disease of the hen) was introduced to the island by poultry imported from the mainland about thirty years ago under the auspices of the Congested Districts Board, and it is said that heavy mortality has been experienced since that date. Through the co-operation of Miss C. Matheson, then Poultry Instructress in Lewis, and later Miss A. MacKinnon, her successor, I was able to obtain an account of this disease and also a supply of dead birds from several outbreaks, and to show that the disease was in fact fowl typhoid.

It is generally considered that fowl typhoid is associated with an unhygienic environment where poultry husbandry is backward and the use of poultry houses with earthen floors is common. The position in certain parts of Lewis supports that view. Poultry keeping is not carried on as a specialised branch

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of farming, and the methods in common use on many of the crofts are extremely unsatisfactory. The farming land is split up into crofts, a number of crofts comprising a village or township. The poultry population per croft may be as small as twenty head, but some carry much larger stocks. In most cases fowls are kept in stone houses with earthen floors, and roam over ground which has been used by poultry for generations, coming into contact with fowls from adjacent crofts or cottages.

The type of house described is difficult to clean and impossible, even if an attempt is made, to disinfect. Little attention is paid to sanitation or the disposal of dead birds, and faulty methods of feeding prevail; all combining to provide an environment ideal for the spread of infectious disease.

Symptoms

There is usually marked loss of appetite, drowsiness, and progressive weakness, accompanied by the passage of liquid fæces, yellowish green in colour.

In some accounts of the symptoms forwarded with bodies for examination there is a history of " whitening of the comb and wattles " (anæmia), whereas in others there is marked " darkening " (congestion) of comb and wattles. This is in agreement with the symptoms generally described.

The period of illness is usually short, and mortality is high. The disease is always more prevalent in spring and summer, and outbreaks are more frequent after rain, due, it is suggested, to the birds drinking from the puddles formed by the rain on the badly drained ground.

Autopsy and Method of Diagnosis

Post-mortem examination usually revealed some degree of enlargement of the liver, which was often congested and dark in colour. The multiple minute areas of necrosis in the liver described in some textbooks were not encountered. In many cases no gross or suggestive lesions were present at autopsy.

Cultures were made from the liver and bone marrow on MacConkey's bile salt medium, and non-lactose fermenting colonies were inoculated into tubes of glucose, lactose, mannitol, dulcitol, maltose and litmus milk, and agglutination tests carried out with the positive sera.

Bacterium gallinarum, the causative organism, forms acid but no gas in glucose, mannitol, dulcitol and maltose. There is a marked late alkalinity in litmus milk, while the organism is serologically indistinguishable from salmonella pullorum, the causative organism of bacillary white diarrhœa.

Control Measures

In view of the geographical situation of Lewis and the almost certain absence of fowl typhoid in birds likely to be imported from the mainland, it

was felt that a good opportunity was presented for testing the efficiency of the measures at present available for controlling and ultimately eradicating the disease.

A scheme was evolved whereby if the disease was suspected birds could be forwarded for examination. If fowl typhoid was diagnosed, all poultry stock on the farm was subjected to the agglutination test. Reactors were culled and half the stock were vaccinated with an autogenous vaccine (killed culture), the remaining birds acting as controls.

The response to the scheme was very poor, and in view of this and also of the fact that only an odd crofter in a community responded, leaving disease centres all around, no deductions could be made regarding the value of the control measures; nevertheless the number of outbreaks diagnosed was sufficient to show that the disease is very prevalent in certain districts of Lewis.

A new scheme has now been arranged whereby it is proposed to confine attention to a given area, in which all crofters are agreeable to take advantage of the scheme and also to assist by the disinfection of their premises.

The selected area involves thirty-nine crofts and is one in which in recent years fowl typhoid has been particularly severe.

It is hoped that by demonstrating the value of preventative measures, other villages and townships may be encouraged to attempt similar eradication measures.

It is of interest that within recent years in certain parts of the island there has been a marked improvement in the poultry stock, and where proper methods of management are in vogue and the fowls kept semi-intensively, fowl typhoid is practically unknown. This is a further demonstration of the important part played by sanitation and management in eradicating the disease.

Summary

A disease known locally as Galair na Cearc, which has been prevalent in Lewis for many years, has been shown to be fowl typhoid.

The backward methods of poultry keeping and lack of proper hygienic measures are of major importance in causing epidemics, as where more modern methods are employed the disease is almost unknown.

A scheme for attempted eradication by means of agglutination tests, vaccination and sanitary measures has been commenced.

Acknowledgments

I would like to express my thanks to Miss C. Matheson and Miss A. MacKinnon, who kindly forwarded notes on the symptoms shown by affected birds and obtained fowls for examination.

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UNIVERSITY TRIBUTE AND PRESENTATION TO EMERITUS PROFESSOR SHARE JONES,

M.D. (V., h.c.), D.V.Sc., M.Sc., F.R.C.V.S.

On Wednesday, December 6, 1939, in the Senate Room of the University of Liverpool.

THE VICE-CHANCELLOR

I HAVE a number of apologies for absence to communicate to you from the Lord Mayor, Mr. R. H. Armstrong, Sir Robert Kelly, Professor H. J. Channon, Professor Henry Cohen, Professor A. Bruce Boswell, Professor L. C. Martin, and I should just like to read a few words from a letter that I have received from Emeritus Professor Hope who says: "My associations with Share Jones have extended over so many years and have been so close, friendly and helpful in the work of the University that it will indeed be a disappointment to me to be unable to attend his farewell function. His long and devoted service in the advancement of Veterinary Science has resulted in many measures of amelioration of the pains and penalties of the animal world. I shall join with you all in your good wishes." I feel it a great compliment that I have been asked to preside at this gathering and to make a presentation to our colleague because I am such a newcomer here, and most of you have known him for very many years indeed, including some who have known him for the whole of his association with the University, which amounts to thirty-five years. I have only known him for two years and one term, and therefore I am merely going to try to give you certain impressions that I have formed of him, although I realise that they are bound to seem to those of his friends who knew him longer a very inadequate statement of our esteem and affection. Now it so happens that Professor Share Jones was almost the first person in this University with whom I was thrown into intimate contact. Up till then (for a reason I am about to mention)—before October 1, 1937, when I took office—I think the only two Professors of the University whom I had met were Professor Abell and Professor Proudman, and for the reason that they both took part in "vetting" me. Incidentally, that word is a very great compliment to your profession, Professor Share Jones, because it has now passed into common usage and when we wish to indicate that a person or a thing has been properly examined, we say that we have properly "vetted" him. You can understand that with Professors Abell and Proudman in mind as the typical Liverpool professors, I got a surprise when, on the occasion, at the beginning of October, 1937, of the visit of the Veterinary Education Committee to Liverpool, there blew into my room a breezy and prosperous North Wales squire. He very quickly took me in hand and we were closeted for many

hours in preparation for the inquiry that was being held by the Veterinary Education Committee. It was Professor Share Jones who initiated me somewhat into the mysteries of your profession, who taught my infant lips how to lisp the language of your science, who showed me how to lead the members of this Visiting Committee to water even if we could not make them drink, who showed me (if I may borrow a word from Mr. Elam's department) how to "restrain" them, and in that way I got to know Professor Share Jones very closely in the first two or three weeks after my arrival in this University. We had a pleasant and interesting time with the Committee, and when eventually we had said good-bye, Professor Share Jones was good enough to come and see me, and it gave me great pleasure when he thanked me for what I had done and told me that I had the love of the animal in my heart. Now if ever there was a man who had the love of animals in his heart, it is the man to whom we are assembled to do honour. (Hear, hear.) I take that as my first characteristic—that Professor Share Jones really does love the animal creation. He does not regard them simply as something to be exploited in the interests of men. There is a remark by Herbert Spencer which is very much in point. He said that "the behaviour of men to the lower animals and the behaviour of men to each other bear a constant relationship." That is eminently illustrated in the person of Professor Share Jones.

Now the second characteristic that I would bring to your attention is the efforts that he has made in the last thirty-five years to raise the status of his profession. Every profession has gone through the struggle of raising itself up from a handicraft to the dignity of a profession. The barber's pole still reminds us that it is not so very long ago that the surgeon was able to cut himself adrift from the barber and become a member of a learned profession, and we have watched in the last two or three decades a very great rise in the standing of the veterinary profession. That has always been, so far as I have been able to judge, in the forefront of Professor Share Jones' ideals. (Applause.) During his career he has contributed to place the profession on a higher basis, by his work on numerous Government Committees and on the Advisory Committee of the Livestock Commission, of which he is now a member, and by his writing and research.

And thirdly, let us remember him as a teacher. When Professor Share Jones first became associated with this University there were—if we exclude the migrants from Edinburgh who were, after all, not really our own flesh and blood—something like twenty to thirty students in this School, and now the Veterinary School is one of our strongest Schools with a complement of one hundred and sixty students, and so steady has been the rise in the type of student admitted to the School that last year all of the students were accepted to read for the degree as well as for the diploma.

The next characteristic that has impressed me is Professor Share Jones' strong civic spirit and his conception of the duty of his profession, not merely

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to concern themselves with the cure of animals and the prevention of animal disease, but to promote the economic welfare of the country by making the best use of the natural resources of the country. It is here that, as it seems to me, Professor Share Jones' political interests and Parliamentary ambitions link up with and derive inspiration from his professional work. There was an occasion some years ago when Professor Share Jones' sphere of activity might have been transferred from Liverpool to Westminster. I understand that he has again placed himself in danger of being transferred from Pentrebychan Hall to Westminster the next time a General Election should occur. I am not supposed to have any politics, and therefore it would be improper for me to express any opinion on the question whether or not we hope he will succeed.

And lastly, I wish to remind you all of his generosity, both in spirit and in action, and his friendship. But for the present circumstances there would have been a much larger body of his friends here to-day, but I have formed the impression in the last two years that when one speaks of Share Jones there kindles in most people's faces a feeling of affection for him. Well now, Professor Share Jones, I shall defer making my actual presentation until Professor Abell and Professor Craig have spoken, but there it is (pointing to the radio-gramophone). You see what it is, and I should like, before I close, to convey to you and my fellow lawyer, Dr. Mary Share Jones, our very warm wishes for long and great happiness together in that house which I have had the privilege of entering, and which I know you love so well, surrounded by the animals that you love so well, and I am quite certain that, just as we shall not forget the services that you have rendered to us, you are not likely to forget the days and years of service that you have spent amongst us. (Applause.)

I will now ask Professor Abell to say a few words.

PROFESSOR ABELL

When I see people here who are much older friends of Share Jones than I, I think perhaps it is almost inappropriate that I should speak. I also feel it rather out of place that I should try to add anything to the words so admirably expressed in the letter to which your signatures are appended. The letter has been drafted by the official phrase-maker of the University whose language may be described as silver-tongued and sincere. The Vice-Chancellor has dealt broadly with Professor Share Jones' work so I need not do so. I find it very difficult as one who has not relinquished his work after thirty-five years in the University to enter into the mind of a man who has. One can only look externally upon his work. One cannot look into his heart and mind. One can see his successes and one can see his failures, but one cannot see what

his hopes and disappointments may have been. At the same time, I am pretty sure that when Professor Share Jones looks back upon the work that he has done in the University he can derive much satisfaction. I know of no one who has seen so much accomplished in his own lifetime. He must realise that he is one of the very few men who have seen their work extend in scope and importance to such a degree. Although I do not want to talk much about his work, I would like to recall his campaign of the middle twenties, and the way in which he fought most bravely and appeared to suffer defeat. As a matter of fact, those defeats have turned out to be victories. He is a man whose ideas for the training in veterinary science were much in advance of many of those in his profession, and were on a much broader basis. If it were necessary to place a seal upon his work, one has only to look at the report of the Veterinary Education Committee appointed by the Ministry of Agriculture to see how his views are supported and the extent to which his recommendations for development have been adopted. In 1925 the majority of the students were people who were only taking the diploma course. As the Vice-Chancellor has pointed out, now, after fifteen years, practically every single man who leaves here has the hall-mark of a Liverpool degree upon him. It is to those men whose calibre is equal to that of the students of any other department in the University that we have to bear the torch of learning. They will bring honour to the profession, to us, and, incidentally, to Professor Share Jones who has encouraged them so much.

I have said what I want to say about his work. Now I want to say something about him. I have been privileged to see Share Jones in his home, and it is not until you see him in his home that you get to know him. I have got several pictures which I would like to portray if I can. You must go into Professor Share Jones' morning room when he and his wife are there. There you will find legions of cats and dogs lying down together. They are all quiet. Occasionally they look up at him with affection because they know him. They know that he loves them and cares for them. You then see where Share Jones' heart is. You see indications of his loyalties to his friends and his devotion to the science which he loves so much. In his pastures you will see the sick, halt and lame; they also look to him for help. Perhaps that is natural.

Or again, you may see Share Jones' majestic figure striding across the top of Offa's Dyke. I can imagine what the famous ex-King of Mercia must have felt when venturing to the Welsh Marches only to find men of Share Jones' stature, vigour, eloquence and fighting qualities confronting him. But those days when the English were fighting against the Welsh have gone. The Welsh have ruled over us for many years, and we of England are proud of the Tudor kings who ruled us and helped build up our characteristics and our traditions. Sometimes the Welsh people forget this. The Welsh have con-

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tributed so much to our national character that I would like them to bear that in mind sometimes.

There is another picture which impressed me very much. You have just to hear Dr. (Mrs.) Share Jones talking about professional matters to realise the important part which she has played in his work. You will realise that she is a woman with keen interest and intellect, intensely interested in him and his work. You will find her always tempering his mercurial spirit and tempestuous energy, guiding here, restraining there, and generally helping him with firm but gentle hands in his work. She, I am sure, has helped the University tremendously in helping him. There is one other picture which I want to draw. It is of a frivolous character, but this gift we are making to him reminds me of it. It illustrates his energy of body and mind. Professor Share Jones gets up with the lark and also keeps company with the owl. Many a night has he dragged me from my bed into my very cold study to listen to his eloquence on veterinary science or politics or agriculture, and his advocacy on behalf of the under dog. On returning home about midnight one night, an evil thought came into my mind. I thought I would ring him up and get him out of bed. When I got through I said: "Look here, you ought to be in bed at this time of night." To my disappointment he said: "I am" (laughter). So I am very pleased to know that he is cultivating, or about to cultivate, one other virtue—he is going to listen. I look forward to the time when I shall see him sitting in front of this machine listening-in.

It gives me much pleasure to convey your best wishes to him and Dr. Mary Share Jones for very many years of health and happiness in their retirement. But neither of these people are of the stuff of people who retire. They will never retire. They will drop in the furrows which they continue to plough to their life's end (applause).

PROFESSOR J. F. CRAIG

Professor J. F. Craig added his tribute to the great personal qualities and the work of Professor Share Jones and associated himself with all that had been so well said by the Vice-Chancellor. He was glad that these tributes should be paid while Professor Share Jones was still with them. Their acquaintance and friendship began while they were still attached to the School in which they received their professional training and had continued uninterrupted outside the University for the most part, but for the last few years as colleagues in the Veterinary School. He could therefore claim that he knew Professor Share Jones for a longer time than anyone in the room. They had many views in common, but although they differed on occasion, their differences were not animated by personal rancour and never interfered with their friendship.

Professor Share Jones has always had a high conception of the value

and scope of veterinary science. Many of his ideas and schemes were not popular when he first gave expression to them, but what one realised was that he had the courage of his convictions and persisted in his views in spite of strong opposition and personal inconvenience. I have always admired him for his courage. He must now be proud in the knowledge that many of his ideas have been generally accepted while others are still the subject of discussion. In these respects he has been a pioneer and has met with and overcome the difficulties of a pioneer.

He came to this University in the first instance because the Veterinary School was to be developed as an integral part of the University, and it is largely through his own energies that the School has attained its present position.

I need only add that I am sure that although he has retired, and his interests may change in other respects, he will continue to look with sympathy and promote the welfare of the University, the Veterinary School and veterinary science.

May I hope that when he is listening to the radio which he is being presented with that when the announcer says: "I wish you well every hour of every day," that wish is ours for him.

THE VICE-CHANCELLOR

It is now my duty to transfer to you formally the possession of this very formidable instrument—a wireless with gramophone combined, and together with it this address which is a very charming attempt to put into permanent form the views of your colleagues towards you and their good wishes.

THE UNIVERSITY TRIBUTE INSERTED IN THE PRESENTATION ALBUM

TO

JOHN SHARE JONES,
M.D. (V., h.c.), D.V.Sc., M.Sc., F.R.C.V.S.

At this time when you withdraw from active participation in that Department of our University where you have spent thirty-five years of your life, we desire to pay our tribute to those qualities of mind and heart which have distinguished your labours here and by which we shall remember you.

We have known your courage, your loyalty, your persistent devotion to a cause and, not least of all, your humanity and kindliness. Your life has been given not alone to the science of Veterinary Anatomy which has represented the field of your professional labours, but also to the arts of healing and of understanding; those quiet and trustful creatures who are commonly called the friends of man have found in you that man can be no less a friend to them.

To one of your restless energy, retirement may not permit leisure, but it

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must surely bring the contentment of battles well fought, of causes devotedly served and of high standards faithfully maintained. And whatever new interests the coming years may contain, we are confident that old friendships will endure; that circle of friendship, of which the University must always remain the focus, includes yet another and most learned graduate in the person of your wife, Dr. Mary Share Jones.

The signatories to the tribute included the Lord Mayor of Liverpool, Pro. Chancellor of University, President of the University Council, the Vice-Chancellor of the University, the Pro. Vice-Chancellor, Deans of Faculties, Emeritus Professors, Distinguished Professors in the Faculties of Arts, Science, Law, Medicine and Engineering and the Schools of Architecture and Veterinary Science, the Acting-Director and Deputy Director of Veterinary Studies, Lecturers in the Veterinary School, together with the past and present Registrars, the heads of the University Administrative Staff, the Secretary of the Board of Veterinary Studies and the Administrator of the Veterinary Hospital.

INSCRIPTION ON RADIOGRAM

PRESENTED TO

JOHN SHARE JONES,
M.D. (V., h.c.), D.V.Sc., M.Sc., F.R.C.V.S.

Professor of Veterinary Anatomy and Director of Veterinary Studies, University of Liverpool, by his friends and colleagues in the University on the occasion of his retirement.

1904-1938.

PROFESSOR SHARE JONES

In his reply to those who had spoken so eulogistically of his services on behalf of the University, Professor Share Jones said that it would not be possible to put into phraseology anything that could adequately represent his innermost feelings just at that moment. " But our minds are inevitably carried back a long time—half the allotted span, thirty-five years, and one naturally becomes reminiscent. We came to this University when the University itself —young, just created—was just blossoming out from birth, full of character, full of characters, men whose names will go down to posterity. I don't think there ever was such an institution as the University of Liverpool so far as that is concerned. One can run through the list of names, and there they stand to-day, they and their work, men congregated in one place, a small affair then, just the end of the preceding year broken off from the old Victoria University and became the University in its own right. Well, we came, an innovation, a novelty, an amusement to quite a large number, into the University. I could

imagine what the Professors of Classics thought when the engineers came some years previously and chimneys and machinery were erected in the University College. What would they say of us who brought horses, dogs and pigs? We were indeed an innovation."

They had no endowment, they had no buildings—not a brick that they could call their own. An old greenhouse that had been converted into a small animal house was lent to them by Boyce and this was converted into a dissecting room.

Reviewing briefly the history of the school in the University, Professor Share Jones said that some of the outstanding features were, firstly the efforts that had been made to establish this division of science on a solid foundation by the endowment of teaching and research positions and giving the holders security of tenure. He regarded this as of vital importance if workers were to put forward their best efforts—more important even than expenditure on buildings which, as Kuno Meyer emphasised, might at any time be burnt down. Taken in conjunction with the older established University positions that were at the School's disposal no other British School was now so well placed as Liverpool in this respect. Not one could be said to be on such a sure foundation; to be entirely dependent upon a Government Grant as Veterinary Schools most frequently had been hitherto, was not in the opinion of Professor Share Jones a satisfactory position in which to place a division of professional education. Furthermore if kept outside the University a branch of education was outside the education stream. His connection with University Colleges in his earliest years had demonstrated this to him and his main object in coming to Liverpool was to do what he could to help to establish veterinary education as an integral part of our general university system on lines similar to those on which other divisions of learning had been established. He now felt that after thirty-five years that had been accomplished in Liverpool (applause); even the youngest member had some security of tenure with corresponding freedom of action in his office.

Then secondly there was the spirit that had been developed amongst the students for University Education and Graduation. At first not a single student was qualified to take a course for a degree had one been available. The new School—established in the face of great opposition, and with much advertisement in consequence—seemed to attract the adventurous type, the type more characteristic of Newmarket, who was endeavouring to get away to Oxford but having nevertheless only just left Newmarket (laughter). Many came to have what they considered to be a good time—some amusement—got it and then left.

The veterinary gathering were a motley crowd not without peculiarities. He recalled a dinner to nineteen new members of the University staff, including veterinary members, when certain happenings prompted him to appeal to those present to suspend their judgment of veterinary men until with the

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effluxion of time that judgment could be based upon actual experience of them when he hoped they would find amongst those engaged in Veterinary Science who devoted themselves to what were termed the lower animals, some imbued with the real spirit of education and disciples of Pestallozzi himself. After thirty-five years he would not hesitate to ask for that judgment now.

They in Liverpool with the sympathy and support of Dale, Hope, Sherrington and others, took a bold step and decided to institute a full degree that would demand a greater expenditure of time, effort, and money than any other similar degree—a degree that would stand comparison with a degree granted to any other profession. To this day it remained the only full veterinary degree granted in Britain. The appreciation of it had been slow but very sure until at present it was reflected in the striking figures the Vice-Chancellor had given. In fact there were more candidates taking the course for this degree than at any other centre in Britain; probably more than at all the other centres combined (applause). Since it was not a licence to practise and therefore not of any direct commercial value they were taking it with the highest possible motive, as evidence of academic achievement. There was no finer spirit than this in any other Faculty or School (applause). Veterinary students who for some years segregated themselves from the rest of the University gradually took their part in University activities and availed themselves of that fuller experience that a University offered. It can truthfully be said that they now make a real contribution to the life and work of the University. There is definite evidence of the part that is being played by both men and women students. There was evidence of it in that gathering (applause).*

A quarter of a century is but a short period in which changes such as these can be effected. They do not come about in a few years (applause).

Professor Share Jones, referring to the University as a whole, expressed his profound gratitude to all his colleagues in the different faculties for the generous help he had always received.

His appreciation of Senate was unbounded—his admiration for the fairness and thoroughness with which all the problems that come before it were considered was great. He assured them that he would miss Senate. There the members were placed in a position of independence with freedom to express their own points of view, a freedom that security in their own positions alone could give them. There all-important branches of learning were or should be represented, and the collective judgment on the points raised secured.

He felt that great as the chief functions of the University had hitherto been in the extension of knowledge by research and instruction in the results secured, it would in the future play an even greater part in our national life. To him it appeared that the freedom of the individual in this country was in

* The two Presidents of the University Students' Union were present and both man and woman Presidents were veterinary students.

some jeopardy—possibly that freedom for which we are engaged in our international struggle. He felt the position of the local governing authorities as representing the individuals was being weakened and the power was being rapidly centralised in Whitehall. Incidentally, this might be in part attributed to the system by which public funds were disbursed as grants. The University would prove a great bulwark of defence and the strongest pillar would be Senate. To achieve a seat on Senate should therefore be the ideal of every University teacher. Conversely, Senate should welcome representation of every division of knowledge worthy of representation. Without this such division would suffer in the place that really mattered and it would be against the University interests. Admission should not depend so much upon finance or the person as upon the subject concerned. This view he had developed as a result of his own experience.

He concluded by paying a warm tribute to the University as an institution. He was grateful to Providence that his feet had been set in the direction of Liverpool. His indebtedness to the associations he had formed there was great and his gratitude for the many benefits he had received would endure unto the end. (Applause.)

He thanked the Vice-Chancellor for his extremely kind and generous speech and references to himself and his wife, to whom he was so much indebted, as also his two colleagues who had spoken, and all his colleagues for the kind manner in which the observations had been received. He thanked them also for their splendid gift and, above all, for the magnificent tribute which they had been good enough to inscribe in the album. His interest in the University would remain.

His wife would wish to be associated with this expression of gratitude and thanks. She regretted her absence, which was unavoidable, but, like his own, her interest in the University, with which she was so intimately connected and with various faculties, would remain. She had never spared herself in endeavouring to advance the cause of veterinary education and he (Professor Share Jones) could endorse all that had been so graciously said (applause).

Pro. Vice-Chancellor Abell's claim for Professor and Dr. Mary Share Jones that "neither of these people are of the stuff of people who retire" may be said to be fully justified, for the Professor is a member of the Court and Emeritus Professor of the University of Liverpool, a Governor of the University College of North Wales, Prospective Parliamentary Candidate for Shrewsbury, Chairman of the Wrexham R.D.C., one of the largest in the country and dealing with half a county, Chairman of the Health, Food Control and other Committees, overseer under the Fuel and Lighting Order, Chairman of the Joint Livestock Committee for North Wales, member of the Government Livestock Advisory Committee, etc., etc., and as is well known to mem-

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bers of the veterinary profession, he is an active member of the Council of the Royal College of Veterinary Surgeons of which he is a Past-President. When Wales became a separate division of the National Veterinary Medical Association he was selected to be its first President.

At the request of the County Council, Dr. Mary Share Jones continues to act as Assistant County M.O.H. and School Medical Officer. A pioneer student of law, she was the first woman to graduate in law at the University of Liverpool, where she took the Degree of LL.B. with first-class Honours.

Clinical Articles

EPISTAXIS

By J. W. HALL MASHETER, M.R.C.V.S.
Ruddle House, Newnham-on-Severn.

SUBJECT.—A four-year-old Friesian-Hereford cow.

On December 3, 1939, this cow was run into "head on" by a motor car, the dumbirons of which penetrated for some distance on each side of the sternum. There was not much bleeding, but a good deal of tissue tear and bruising. The wounds were dressed and plugged.

December 4.—Brisket very much swollen and painful. The swelling interfered with locomotion. Wounds syringed out, replugged and 10 c.c. Yatren vaccine E 104 given subcutaneously. Progress was good until:

December 15 when she was reported to be bleeding very strongly from the left nostril. I was not able to get to the farm for a couple of hours, and when I did arrive the loose box was like a slaughterhouse—blood all over the floor, in the channel and across the yard. It was arterial blood from the left nose somewhere, but was too high up the septum for me to locate it. There was no evidence of injury either externally or internally.

I attempted to plug the septum, but this only complicated matters, as it forced the blood back into the fauces and set up coughing and choking—the plugs were sneezed out time after time, so I gave up that method of stanching nasal bleeding.

The cow had lost a considerable amount of blood, but was not really weak, only (for her) subdued. Three cubic centimetres of adrenalin, 1-1,000, were then injected intramuscularly. In about ten minutes the stream of blood was decidedly reduced in volume, and in 30 minutes had stopped, only to recur

three hours later. Another 3 c.c. again stopped the bleeding. On December 16 in the early morning another message to say that the bleeding had recommenced. Another 3 c.c. adrenalin stopped the bleeding. I saw her again late afternoon, but there had been no recurrence of the hæmorrhage. I then gave 3 c.c. adrenalin subcutaneously as I thought absorption would be slower and would keep up the effect, which it did. The cow "made an uneventful recovery."

The query here is: What started nasal hæmorrhage 12 days after the accident?

Abstracts of Current Literature

Egg Counting Technique for Use in the Diagnosis of Equine Strongylosis. By D. W. Baker and J. W. Britton (1939). Report of the New York State Vet. College, No. 18, 175.

THIS paper is an account of methods of estimating the actual number of strongyles derived from adult worms and deductions are made as to the best time to treat horses with anthelmintics.

Two estimations were carried out at intervals over a period—actual egg counts estimated as eggs per gram of fæces and the number of larvæ developing from the eggs—estimated by deep plate cultural methods. By carrying out post-mortem examinations the number of the various kinds of female strongyles was found and from the different bits of information it was calculated that the average rate of egg production per adult female worm was: S. vulgaris, 1.79, S. equinus 1.78, S. edentalis 2.1, small strongyles 0.5-1.0. (The calculation made was as follows: e.g., 2,150 eggs per gram of fæces. Larval count = 5% S. equinus. P.-M. examination showed 65 female S. equinus. Thus $\frac{2{,}150 \times 0.05}{65} = 1.65$ eggs per gram per adult female S. equinus.)

Various factors influencing the development of larvæ and the further stages of the worms are discussed and the final conclusion is that the spring months are best for the most effective treatment of horses for strongylosis since

fewer larvæ would be migrating in the tissues and anthelmintics would then be able to deal with the adults. Various fluctuations in counts, etc., were noted, some of which could not be accounted for.

The Gestation Period in the Bitch. By L. F. Whitney (1940). *Vet. Med.*, XXXV, 59.

The author has studied this subject largely in his own kennels. Following many hundreds of matings, from which over 5,000 puppies were born, only in 30 instances when mating was allowed on one occasion did pregnancy result. The paper deals largely with these 30 bitches.

Gestation is defined as the period elapsing between the fertilisation of the ovum and the birth of the puppies. It cannot, however, be regarded as the period between coitus and birth, for it is known that the ovum can exist several days in the reproductive tract of the female before fertilisation occurs. It is accepted by the author for the purpose of this paper that ovulation occurs about the fifth day after œstrus is observed and he bases his conclusions on a survey of the period elapsing between matings near the ovulation time and birth.

He had already shown in another paper that the sperm reaches the ovarian bursa by the time copulation is completed and that fertilisation soon occurs. The fertilised ova has now only to traverse the short Fallopian tubes and move down the short horns of the uterus where they become implanted and start to develop. (It is noted that the ova may already be in the uterus where fertilisation will take place.) If copulation has taken place prior to ovulation the sperms must live in the tract for some time and thus the gestation period will probably be longer.

Of the 30 cases studied, in 13, where coitus took place before the fifth day, the average length of the gestation period was 64 days. In the other 17 cases copulation occurred on or after the fifth day and the average gestation period was found to be 60.7 days.

The author has some further observations to make in a later paper. He has evidence to show that a bitch bred to two genetically dissimilar dogs at four days' interval produced puppies sired by both dogs (five by one and six by the other). From the first day of mating by the first dog till birth, 63 days had passed and in the second dog 59 days.

From all the evidence in the possession of the author he concludes that the gestation period in the bitch is about 61 days, with some variations.

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Diaphragmatic Hernia in Equines, not of Traumatic Origin. By B. F. Trum (1939). *Vet. Bull. supplement to the Army Medical Bulletin,* XXXIII, 297.

Details of three cases are recorded.

The first horse was found sick and died within 17 hours. The post-mortem examination showed a rupture five inches long in the tendinous portion of the left part of the diaphragm under the lumbo-costral arch. The edges were smooth without evidence of hæmorrhage. Several inches of the small intestine had passed through into the pleural cavity.

The second case was that of a 12-year-old mare which died within six hours of being reported ill. Among other things found at post-mortem examination was a rupture in the diaphragm about 14 inches long. All but four inches was definitely a post-mortem rupture—the remaining four inches appeared to be long standing.

The third case was a four-year-old gelding which was found suffering from colic. The symptoms suggested a case of strangulation, obstruction or some such intestinal derangement. Peristalsis sounds were detected over the thorax though it could not be ascertained that they were actually heard within the pleural cavity. Traction was applied to the part of the small intestine within reach. A sudden releasing of tension was felt and respiration, which was fast and shallow, immediately became normal. The animal was discharged cured next day and the author holds the opinion that in this animal was a diaphragmatic hernia.

The author discusses the cases and refers to the development of the diaphragm, and concludes that in equines, especially young ones, the possibility of a diaphragmatic hernia cannot be considered too remotely when diagnosing cases such as he records. He believes that a portion of the intestine may pass through the hernia without causing much discomfort to the animal provided twisting or strangulation does not occur.

Nutritional Deficiency in a Litter of Great Dane Puppies. By A. R. Campbell (1939). *Canad. Jl. of Comparative Medicine,* III, 288.

A five-week-old Great Dane puppy was presented to the author for treatment for worm infestation. He formed the opinion that it was suffering from a nutritional deficiency of an acute nature. The symptoms were extreme

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prostration, the puppy being unable to stand, nervousness, irritability, sensitiveness and pain shown by persistent muscular twitchings and continual howling and whining. A subcutaneous injection of 10 c.c. of a 20 per cent. calcium gluconate was given. Four days later the whole litter of five puppies were brought for examination. They had been weaned ten days and all of them seemed to be affected similarly to the first puppy, though not so severely. Advice as to feeding was given—bonemeal and bran were added to the diet and plenty of milk and brown bread was included as well as cod liver oil. Three weeks later the owner reported that all the puppies were doing well. The author states: "It may be timely to remark that in the larger breed of dogs (the young of which grow quickly) it is necessary to give special consideration to the feeding of the dams while they are pregnant and also while nursing their young. When the puppies are weaned they should be provided with a ration adequate and sufficiently varied in character to meet with the demands of growth and development."

Domestic Fowls as Hosts of Poultry Gapeworms. By E. E. Wehr (1939). *Poultry Science,* XVIII, 432.

This article is an account of some experimental work in which birds reared under parasite-free conditions were infected experimentally with gapeworm (Synganus trachea). They were fed with eggs and larvæ obtained by culturing the eggs of female worms removed from the tracheas in infected turkeys. Evidence of infection was obtained from fæcal examinations of the birds under experiment. It was shown that the guinea fowl, the young chick and the turkey are important hosts of gapeworms. In the pigeon it would appear that the larvæ reach the lungs, develop to their fourth stage and then die.

The author considers that the results of his observations in pigeons "suggest a strong host reaction resulting in the destruction of the larvæ." Ducks (the Pekin variety only) were also used and it was shown that they were not a suitable host for the parasite. Concerning the duration of the infective period following administration of the above material, the evidence shows that in chickens it may be as long as 147 days, in the guinea fowl, 98 days and in the turkey 224 days. It is stated that "evidence is available for the first time to show that the turkey may be responsible for the perpetuation of gapeworm disease from one warm season to another."

Canine Leptospirosis in the United States. By K. F. Meyer, B. Stewart-Anderson and B. Eddie (1939). *J.A.V.M.A.*, XCV, 710.

This paper is a valuable contribution to the subject of canine leptospirosis. It will be remembered that in 1925, Okell, Dalling and Pugh described infection in dogs with L. icterohæmorrhagica in this country and showed that it was the cause of "yellows" and that Klarenbeek and Schüffur recognised L. canicola as a cause of leptospirosis in dogs in Holland in 1934.

The authors of this paper carried out their observations in California and record full details of their methods and conclusions. For diagnosis purposes they made use of three types of tests—the lysin-agglutination test; blood cultures and guinea-pig inoculations with blood; urine examinations by dark ground, serological procedures, cultures and animal tests.

There existed among their cases two distinct types of disease—one, in which jaundice is present and the symptoms and post-mortem appearances resemble closely "yellows" in this country, and the other, the hæmorrhagic type, which resembles canine typhus or Stuttgart disease. The symptoms and lesions are described in detail.

Dark ground examination is criticised and it is pointed out that, as those familiar with this type of work are aware, the presence of mobile filaments in wet preparations may lead to false conclusions.

Until recently it appeared that all leptospira infections in California were due to L. canicola, irrespective of the train of symptoms and lesions observed, but recently there is evidence from serological study that L. icterohæmorrhagica infection may also be present. Up to date no specimens of the latter have actually been recovered from dogs examined by the authors. (This is in striking contrast to the results in this country.)

L. canicola was found commonly in the kidneys of infected dogs, either singly or in clusters, as seen in sections, and their presence in the urine is therefore to be expected. Infected urine is probably the chief source of infection.

It is striking that the disease associated with icterus is connected with L. canicola and not with L. icterohæmorrhagica as occurs in this country and in Holland. It is pointed out that mistakes in diagnosis may occur in the hæmorrhagic type of the disease because of the belief that all cases of black-tongue in dogs are due to a dietary vitamin G deficiency; the authors state that there is an increasing number of reports of so-called blacktongue in cities and towns where the diet is rarely a deficient one.

The authors recall that the Dutch veterinarians have observed a form of chronic leptospirosis predominantly characterised by a nephritis, high non-protein content of the blood and uræmia. Although this type has not been found by the authors they believe that it may well occur and suggest that systematic examinations of urine for leptospira may disclose its existence.

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It is further suggested that many atypical and abortive illnesses may be sub-clinical or latent leptospirosis and that such infected dogs may be sources of infection to others dogs and to human beings. (It is interesting to observe here that the authors failed to demonstrate L. canicola in rats, the vectors of L. icterohæmorrhagica. They conclude that the rat is not a natural host for the former.)

The use of specific antiserum in the treatment of the disease is recommended.

There is an extensive bibliography and in three appendices methods of staining, culturing and serum testing by the porcelain-plate agglutination method are produced in detail

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A Survey of Tubercle Bacilli Types and of Type Diagnosis. By H. Holth (1939). *Skand. Veterinär-Tidskrift,* XXIX, 1125.

The author acknowledges the difficulty of demonstrating experimentally that one type of tubercle bacilli becomes transformed to the other, though he gives several examples that such may have occurred.

Concerning diagnosis of the various types he relies on animal inoculation and culture work. He states that the human type inoculated into guinea-pigs causes progressive tuberculosis but in rabbits the results are negative or develop very slowly, while in fowls, entirely negative results obtain. The bovine type causes progressive tuberculosis in both guinea-pigs and rabbits but produces no result in fowls. The avian type produces no results in rabbits or guinea-pigs but in fowls the progressive disease develops.

As to culture work, the author uses Petragnani's medium. Cultures of the human type are thick, dry, rough and yellow with large, irregularly limited, isolated colonies resembling breadcrumbs or a cauliflower. With the bovine type the culture is a fine, hardly visible film with yellowish-white, shiny colonies, about the size of a pin point. The avian type produces grey, dirty white or yellowish moist or mucous colonies; the isolated colonies resemble small dots.

The author has devised a new medium for use in typing cultures. It consists of an egg and potato medium to every 100 c.c. of which are added 2 c.c. tuberculin and 3.35 c.c. of 5 per cent. lactic or hydrochloric acid. It is claimed that on this medium sub-cultures can be obtained in 3 to 4 days after sowing.

An Avian Semen Collector. By J. E. Parker (1939). *Poultry Science,* XVIII, 455.

This contribution comes from Missouri Agricultural Experimental Station. A special piece of apparatus is described and illustrated and details of its fixation is given. It is stated that males from which semen is to be collected should be separated from the females for 12-48 hours and that the risk of contamination should be reduced by removing feathers from the vent and cleansing the region. When the apparatus has been placed in position the bird is allowed the freedom of the breeding pen. After each mating the semen is removed from the collecting flask of the apparatus by means of a glass syringe.

Artificial Insemination in New York State Dairy Herds. By G. E. Burch (1939). *Cornell Vet.,* XXIX, 395.

This article is a record of experiences in the artificial insemination of dairy cows and has been written primarily for the benefits of veterinary practitioners.

The evidence would show that the average bull cannot stand service oftener than once every three days over an extended period and still deliver a satisfactory ejaculate, especially if the semen is to be used artificially. Storage of semen is discussed. The results of experiments are somewhat conflicting. Selected samples of stored semen when used 24 or more hours after collection compared favourably in fertility with average fresh semen samples. Some bulls, however, provided semen which was quite inactive after storage and, further, all samples from the same bulls did not behave similarly after storage.

The effect of artificial insemination on the occurrence of abortion is dealt with but, though of 336 pregnancies resulting from artificial insemination, only 7 ended in early abortion, the results are of limited significance for the author notes that a sufficiently long time has not yet elapsed to make definite statements and no comparisons could be made with normal breeding results.

The author found that animals bred early in heat and those bred late in heat are less likely to conceive than those bred at 18-14 hours from the onset of external symptoms of œstrus and that virgin heifers conceived more readily than mature cows.

A detailed description (of much value to the practitioner) of the technical procedure is given and many valuable hints are included.

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The Efficacy of Sero-Vaccination of Lambs Against Tetanus. By C. W. Adey, D. T. Oxer and May Kennedy (1939). *Aust. Vet. Jl.*, XV, 205.

Sero-vaccination means a simultaneous injection of tetanus antitoxin and of tetanus toxoid. The experiments undertaken by the authors were to ascertain whether these substances, introduced simultaneously, would confer both an immediate and a lasting immunity. The importance of these experiments is considerable for although antitoxin will certainly give an immediate protection against tetanus following castration, etc., the immunity is short, and the lambs are soon again susceptible to later infections.

Eighteen lambs were divided into three groups—A. B and C. After estimation of the initial tetanus antitoxin contents of the blood of each lamb, group A was injected subcutaneously with 100 units and antitoxin; group B with 1 c.c. tetanus toxoid (alum precipitated); group C with both products, one into each thigh. Titrations of the antitoxin content of the blood of all the lambs were carried out at intervals over a period of 65 days following the treatment. The results were:

Group A.—The quantity of antitoxin decreased progressively for three weeks and then could no longer be demonstrated.

Group B.—Antitoxin appeared on the 8th day then rapidly increased and was continually present during the test period.

Group C.—Antitoxin was found throughout the whole of the period. For the first three weeks the level corresponded to that in Group A and remained more or less unaltered throughout the experiment; it never rose, however, to the high level in Group B.

The same 18 lambs were now tested for resistance to tetanus infection. Spores activated with calcium chloride were injected. The time interval between the original treatment and the test varied from 99 to 135 days. At the 99 day period, the lambs from group A died while those from B and C remained well. At the 109 day period, two lambs from each group were tested—all except one from group A remained well. At the 121 day period double the dose of spores given to the above lambs was used on three more lambs from each of the groups. Here again one of group A lambs died. There now remained 15 lambs (all already treated). They were all tested 135 days after the original treatment, a massive dose of spores being used. All the lambs in group A developed tetanus while those in group B and C remained well.

The experiment shows that the simultaneous injection of tetanus antitoxin and tetanus toxoid (alum precipitated) into lambs affords both immediate and lasting immunity as shown by artificial infection, though the lasting protection is not of such high order as that arising from the same dose of the toxoid when injected alone.

News

ALAN, DUKE OF NORTHUMBERLAND MEMORIAL FUND

SIXTH REPORT OF THE MANAGEMENT COMMITTEE, 1940

THIS report, prepared by W. Lyle Stewart, M.R.C.V.S., King's College, Newcastle-upon-Tyne, is largely an account of research work into sheep and lamb diseases.

In the foreword it is stated that the object of the Fund, in addition to commemorating the public services of Alan, eighth Duke of Northumberland, is to assist "investigational work into the more obscure diseases of livestock, with a view to bringing them under control." The technical work of the Fund is carried out by members of the staff of the Veterinary Branch of the Agricultural Department of King's College, Newcastle-upon-Tyne, under the leadership of W. Lyle Stewart. The report can be divided into three sections: (a) distribution of biological and other products, (b) sheep and lamb diseases (notes on common preventable diseases), and (c) research articles by W. Lyle Stewart and A. Phyllis Ponsford.

The Fund, acting in conjunction with the North of England Veterinary Medical Association, issues to its members vaccines, serums, etc., at reduced cost. As an example of the amount of material handled, it is stated that in 1939 over 50,000 doses of lamb dysentery products and over 100,000 doses of nicotine mixture were issued. To the list of products available to members have now been added louping-ill vaccine and Northumberland tick powder, while it has been found necessary to discontinue the issue of nicotine mixture. Field trials are still to be made in 1940 with pustular dermatitis vaccine and a treatment for "moss staggers."

The part dealing with sheep and lamb diseases portrays in simple language the salient facts concerning the symptoms, etc., and control of diseases such as lamb dysentery, pulpy-kidney disease, braxy, louping-ill, swayback, etc.—all of which are diseases found in the district concerned.

The section concerned with research articles is of special interest to the veterinary profession. One article deals with *the control of sheep ticks*. It is pointed out that wide areas in the Northern Counties of England are tick infested and that much damage is done to lambs on the farms in the area by ticks. Chief among the diseases caused by ticks are: (1) septic infections (pyæmia), resulting from the tick bites, and (2) louping-ill, the causal virus

being conveyed by the tick. The investigations carried out by Lyle Stewart and his colleagues showed that the application of powdered derris root was effective in keeping ewes tick-free better than the best dipping solutions they tried. The preparation was also effective in preventing tick infestation in young lambs and its application reduced lamb mortality.

Border pining is also discussed and in its investigation the Veterinary Laboratory of the Ministry of Agriculture has also participated. It was shown that both worm infestation and some nutritional deficiency are involved in the causation of this disease. Up to the present the workers have been unable to show that a deficiency of cobalt *per se* is the cause, though it is stated in the report that combinations of cobalt and other minerals have yet to be tested. Investigations such as those carried out under the Fund are certainly altering some of the accepted and commonly practised customs in the district. The following extract from the report is significant: "Hill farmers may be surprised to learn that blackface sheep can be confined to a limited range of rough hill grazing in a pining area for three years, without change of pasture, if dosed at three-weekly intervals with nicotine mixture or if they are given a small allowance of cake."

Lastly, the latest results of research into pregnancy disease of ewes ("staggers") are reported. It is shown that two types of "staggers" exist—"moss staggers" which occur in hill ewes and with which is associated a low blood calcium content, and "pregnancy toxæmia," in which acidosis is present. Recommendations for treatment and prevention are made and the assistance of farmers is solicited to test them.

The report certainly shows that the workers are carrying out a large programme and they are to be congratulated on their efforts to be of practical assistance to the sheep farming community in the Northern Counties in particular.

MINISTRY OF HEALTH

KEEPING OF PIGS AND POULTRY ON LOCAL AUTHORITIES' HOUSING ESTATES

THE Minister of Health, in a circular to housing authorities, urges that in view of the importance at the present time of increasing the food supplies of the country, they should consider how far they can reasonably remove or

relax any existing restrictions on the keeping of poultry and pigs by tenants on their housing estates.

While recognising that the decision rests entirely with the local authorities, the Minister expresses the opinion that there are few places where poultry-keeping might not be permitted without detriment to health and amenities, provided that proper safeguards are imposed. These safeguards should include the use only of types of hen cote approved by the local authority; control over the siting; restriction, according to the space available, of the number of birds kept by individual tenants; and a ban on the keeping of male birds of such an age as to be capable of creating a nuisance to neighbours by crowing.

As regards the keeping of pigs by householders, any local by-laws are normally in a form which, subject to cleanliness, does not limit the distance from a dwelling within which pigs may be kept, though certain specific requirements are laid down in the interests of health and to obviate nuisance. Some local authorities, however, impose more onerous conditions on the keeping of pigs by tenants of their houses or even prohibit it entirely. The Minister hopes that these authorities will in present circumstances be ready to modify or remove any such restriction as far as is reasonably practicable.

TUBERCULOSIS IN PEMBROKESHIRE

At a meeting of the Pembrokeshire County Council Contagious Diseases Committee (as reported in the *Swansea Guardian* of December 15, 1939) tuberculosis in cattle was discussed following the presentation of the quarterly report by the Ministry of Agriculture's Divisional Inspector for the County. It was pointed out that there were now 863 attested and 671 supervised herds in the county.

Criticism was made by one member of the decision that no fresh herds were to be taken for the attested herds scheme and a resolution was moved and carried urging the continuation of the attested herds scheme.

The incidence of tuberculosis in the county was extremely low—573 herds, comprising over 19,000 cattle, had been inspected and only one case had been dealt with under the Tuberculosis Order.

The disposal of reacting cattle was criticised, a member remarking that they were sent to the marts and so found their way into other herds, it being suggested that all reactors should be destroyed.

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OBITUARIES

SIR JOHN MOORE

JUST as we were going to press the sad news was received of the death of Major-General Sir John Moore, K.C.M.G., C.B., F.R.C.V.S. He was director of Army Veterinary Services in the last war. We hope to publish an appreciation of Sir John Moore in our next issue.

MAJOR-GENERAL W. D. SMITH

BY a curious coincidence the death was also announced on the same day of Major-General W. D. Smith, C.B., C.M.G., D.S.O., M.R.C.V.S., who also held the position of director of Veterinary Services and, until his retirement in 1921, was Director-General of Army Veterinary Services at the War Office.

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